SURGERY, GYNECOLOGY AND OBSTETRICS

AN INTERNATIONAL MAGAZINE, PUBLISHED MONTHLY

VOLUME 60 MAY, 1935 NUMBER 5

SEQUENCES OF EXPERIMENTAL BACTERIAL INFARCTION OF THE FEMUR IN RABBITS

GENE H. KISTLER, M.D.,[1] UNIVERSITY, ALABAMA

INFARCTS were produced in the femurs of rabbits by particulate suspensions of charcoal and the sequences in such lesions were correlated with a discussion of some of the more obscure clinical conditions which essentially are focal necrosis of bone marrow tissues (17). The experiments reported demonstrate that infarcts occur in bone in spite of its great vascularity and abundant blood supply, and that the metaphyses are more vulnerable than other regions. Since the embolic substance, charcoal, is extraneous and was injected in relatively large amounts, experiments, simulating more nearly the conditions occurring spontaneously in bone, were undertaken with dead and living bacteria as emboli. By these procedures it seemed possible to produce septic and aseptic necroses of bones where a circulation disturbance (infarction) is the common factor, and thus offer an explanation why these lesions occur so frequently in certain locations.

For many years it has been known and generally accepted that osteomyelitis, not directly the result of injury, tuberculosis and various forms of so called aseptic necrosis and osteochondritis, occur almost exclusively in the epiphyses of bones and the metaphyses of growing bones. The essentials in the production of these lesions and a satisfactory explanation of their occurrence in special regions, more often in certain bones, have aroused considerable interest, experimental investigation, and speculation. The evidence available favors that a nutritional disturbance is an important factor in some, and infection in others, but there is no accepted concept of the details involved.

Lexer and his associates stated that the smaller branches of the nutrient artery of the shaft do not pierce the epiphyseal plates and that the resulting hyperæmia and sluggishness of the blood stream favor the deposition and retention of bacteria in the metaphyses. According to Axhausen, Koenig and many others, occlusion of one of these terminal vessels may produce infarction of the tissues. This is the most prevalent theory of how hæmatogenous foci of osteomyelitis occur. Wilensky stated that thrombosis, following embolic occlusion of vessels, is an important factor. Phemister (29, 30) believed that such an embolus must be infected to produce noteworthy necrosis. According to Wakeley the blood vessels at the actual margin of the metaphysis are in a series of narrow and inelastic tortuous loops, an arrangement which favors embolic infarction. Schulze noted that the metaphyseal capillaries have an elongated hairpin shape, while the subchondral epiphyseal capillaries form a coarse network with nodular projections toward the cartilage. He assumed that in the elongated capillaries the blood current is slow and that there are no protective cells of the marrow, since India ink injected into young dogs was

[1] John Jay Borland, Fellow for Clinical Research, St. Luke's Hospital, Chicago. From the Henry Baird Favill Laboratory of St. Luke's Hospital, Chicago, and the University of Alabama Medical School, University, Alabama.

absorbed more slowly from the metaphyses than from the diaphysis tissues.

According to Kolodny, Eliason and Ferguson, and Harbin and Zollinger, one or two small vessels pass from the diaphysis to the epiphysis through the conjugal cartilage but have little significance. Harbin and Zollinger stated that the line may also be penetrated by vessels in rickets and in some other pathologic conditions. Harris (13) demonstrated by cleared preparations previously injected with carmine-glycerine, that diaphyseal vessels do not pierce the cartilage plates to enter the epiphyses. Vogt (1878) and Jahn (1892) demonstrated that mechanical disturbance or removal of epiphyseal cartilage plates interferes with growth of the long bones. More recently, Meisenbach, Haas, Bohlman and others found that various chemical and solid materials placed within the metaphysis decrease the longitudinal growth of bone and that transplants of epiphyseal cartilage do not produce bone. They emphasized the need of a metaphyseal blood supply for osteogenesis. According to the clinical observations of Sever, separation of the epiphysis usually takes place between the metaphysis and the epiphyseal cartilage plate and as a result of the anatomical arrangement there may be no gross interference with the circulation or viability of the epiphyseal cartilage columns. Occasionally, authors have hypothecated the presence of physiological end-arteries in the growth centers.

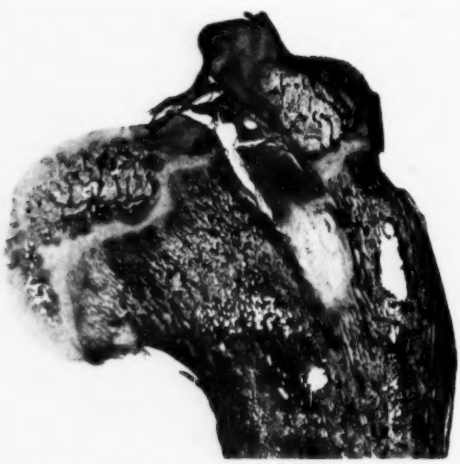


Fig. 1. Marked acute aseptic necrosis of the head and neck and medial bending of the proximal end of a femur 4½ days after infarction by agglutinated killed staphylococci. ×7.

Since osteomyelitis is so much more common in children, particularly boys, and often in the extremities, external trauma always has been considered a factor in localizing such a focus. Likewise, trauma to the articular surface and subchondral bone tissues of epiphyses is thought by many to be responsible for the various forms of osteochondritis. Such injuries as these are not similar to the trauma of weight-bearing during which coxa vara deformity at the hip is produced or an epiphysis is slipped, resulting from some underlying alteration in the structure of the bone or cartilage. According to Reischauer and his statistics, trauma probably is of no importance in the origin or localization of osteomyelitis or tuberculosis of bone. He considered that these diseases attack the metaphyses because of active cell proliferation and tissue metabolism in certain metaphyses more frequently than others because of a greater rate of growth.

In an extensive review of necrosis in different bones, Harbin and Zollinger correlated these diseases, often referred to by various specific names, into one group known as osteochondritis of the growth centers. They believed that the etiology of these is unknown but that direct trauma or indirect as a result of increased stress and strain from the body forces, seem important, while heredity may be a factor. Wardle stated that slipping of the head of the femur may be due to localized osteomalacia which, tending to occur in the areas of most recently formed bone, weakens the attachments of the epiphyseal cartilage. In his discussion of tuberculosis of bone, Oberzimmer emphasized the resemblance of these cuneiform regions of necrosis in articular extremities to infarcts. These foci are subchondral and usually triangular, the base toward the articular surface. He concluded that the genesis of these necrotic regions is not understood and if they are true infarcts, emboli in some of the vessels or an obliterating endarteritis should be found.

In his discussions of silent foci of osteomyelitis and chronic fibrous osteomyelitis, Phemister (31, 32) stated that these lesions often are metastatic, remain symptomless for a long time, and usually occur near the ends of bones. He said that the etiology of these lesions is not known; neither do we know the relation of chronic fibrous osteomyelitis to bone cysts, osteitis fibrosa cystica, giant cell tumors, and pyogenic osteomyelitis. According to his views, embolism and infarction may play a rôle in the production of some of the lesions, but pyogenic bacteria of low virulence must also be present, because some of the cases of chronic fibrous osteomyelitis reported could not be explained on an aseptic necrosis basis alone. Compère cultured a green-producing streptococcus from a lesion in the astragalus that histologically was similar to osteitis fibrosa. Greenwood, Pease, Phemister, Brunschwig and Day (33), and many others have isolated various organisms from patients with Koehler's, Kienboeck's, Osgood-Schlatter's, and other related diseases, which often are placed in a group as aseptic necroses or osteochondritis of individual bones. According to Hallermann, arteriosclerotic changes of the nutrient blood vessels of the upper end of the femur are often associated with osteoporosis of that portion of the bone. It seems that practically all diseases of bone, except those attributed to dietary or glandular disturbances, have characteristics in common which probably are associated with peculiar structural arrangements of the subchondral tissues that make them more vulnerable than other bone tissues to disturbances in blood supply. The individual differences among these diseases, therefore, may be based upon the presence or absence of infection, and if infection is present, upon the mass and virulence of that particular organism.

Experimental studies have aided little in understanding fully the infection and necrosis of bone tissues. Senn refers in his text to experiments by Struck, Krause, Mueller, and Rodet who injected animals intravenously with pyogenic organisms, usually staphylococci isolated from osteomyelitis, and noted acute suppurative lesions in bones, joints, and many other tissues. They often found the foci in bones of young animals near the epiphyses without accompanying fracture or other external evidence of injury. Mueller also produced in young animals, a few characteristic foci by injecting tuberculous material. Recently Schulze observed similar results which support the view of Lexer concerning the mechanism of localization of foci in bones. Jackson noted wedge-shaped regions of necrosis in the metaphyses of bones during an attempt to produce arthritis in rabbits by the intravenous injections of streptococci. From his studies of experimental osteomye-

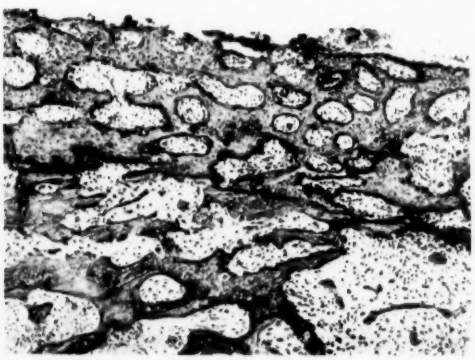


Fig. 2. New bone (involucrum) formation which took place outside the devitalized cortical bone near the proximal end of a femur 8 days following infarction caused by the injection of the agglutinated dead staphylococci. ×36.

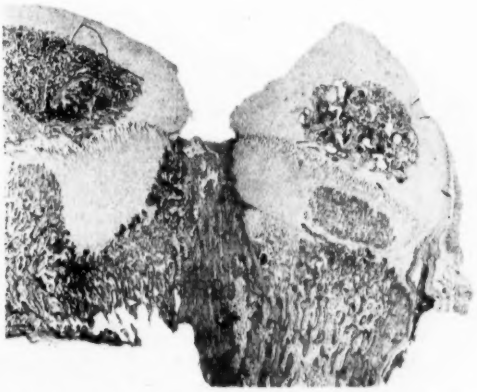


Fig. 3. Marked hypoplasia of ossification in the epiphyseal cartilage beneath the head and great trochanter of a femur 8 days after infarction by agglutinated killed staphylococci. Beneath the cartilage plate of the trochanter is a so called line of arrested growth. ×10.

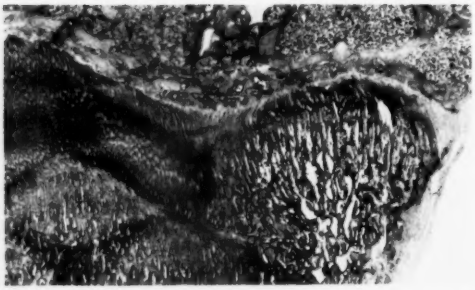


Fig. 4. Marked overgrowth of the central portion of the distal epiphyseal cartilage 18 days after infarction of the metaphysis by agglutinated killed staphylococci. The peripheral portions of the plate received sufficient blood for ossification from the periosteum. ×11.

Fig. 5. A different level of the metaphysis in Figure 4 demonstrating considerable distortion and scarring of the epiphyseal cartilage plate. Collateral circulation and subsequent ossification have been established from the epiphysis but these extend only to the necrotic tips of the cartilage columns. ×11.

litis in dogs, none of which lived longer than 48 hours. Starr concluded that infection starts in the metaphyses, extends along the epiphyseal line to the cortex and periosteum, and probably spreads along the shaft through the Haversian canals. Kuwahata produced abscesses in bones of young but not of adult guinea pigs by injecting a strain of Staphylococcus aureus into the heart. The incidence of the abscesses was much greater when the diet of these animals was deficient in vitamin C. He suggested that a peculiar vascular arrangement in the metaphyses and less resistance of the tissues than elsewhere may play a part in localization of the foci. Haldeman and others studied the acute osteomyelitis in animals produced by injecting a culture of a pyogenic organism through an opening in the cortex. Such experiments do not repeat the conditions with spontaneous lesions and do not aid in understanding why pyogenic infections occur in certain regions of bone. It seems there are no controlled observations of lesions produced in bones where living and dead bacteria have been used as emboli, thus determining any relation between bland or septic infarction to focal necroses of bone. Also, the exact location and tissue structure

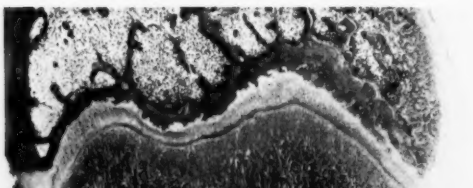


Fig. 6. Early diffuse septic necrosis in the distal metaphysis of a femur 16 hours after injection of agglutinated living cocci. There is a distinct line within the cartilage plate which indicates the level of potential epiphyseal separation. ×7.5.

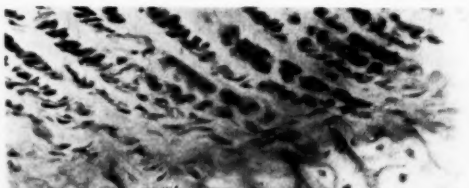


Fig. 7. Masses of living staphylococci wedged in the lacunar spaces between the columns of calcified cartilage matrix of Figure 6. These denote the location of abscess formation in the metaphysis. The extent of necrosis in the epiphyseal line is limited sharply. ×221.

Fig. 8. Wedge-shaped acute metaphyseal subchondral abscess of the proximal end of a femur 2 days after receiving agglutinated living organisms in a suspension of charcoal. There is beginning separation within the epiphyseal line. ×16.

Fig. 9. Multiple subchondral abscesses of the great trochanter and marked necrosis in the metaphysis of the femur in Figure 8. There is beginning liquefaction within the epiphyseal cartilage plate. ×9.

of early lesions have not been studied in detail, either clinically or experimentally in animals.

EXPERIMENTS

A hæmolytic strain of Staphylococcus aureus, cultured from the blood stream of a man with a subperiosteal abscess and fatal septicæmia, was injected through the principal nutrient arteries of the femur of rabbits by the method used for infarction of this bone by a particulate suspension of charcoal (17). To agglutinate the organisms and partially or completely sterilize the suspensions, from 0.1 to 0.4 cubic centimeter of one-tenth normal sulphuric acid were added 2 to 48 hours before injection. The suspensions were obtained by washing 24 hour agar slant cultures with 1 cubic centimeter of normal salt solution. The degree of agglutination, usually marked, was demonstrated by gross and microscopic examinations and cultures of the suspensions were made at the time of injection. It was thought that mildly septic infarcts compatible with life could be produced by decreasing the number and virulence of organisms acting as emboli.

Rabbits between 3 and 8 weeks of age were chosen in order to study the localization in active growth centers of bland and septic emboli. The agglutinated cocci were forced into the chief nutrient artery of the shaft by retrograde injection through the femoral artery with other outlets temporarily ligated. In a similar way, the suspension was directed through the deep femoral artery, one branch of which enters the trochanteric notch of the femur, another encircles the capsule of the hip joint, and a third becomes the principal source for the vessels in the ligamentum teres. Because of the small caliber of the latter vessels, exact and well controlled isolated infarction of the head of the femur was not possible. Material introduced into the principal nutrient artery of the shaft was directed almost entirely to the medullary canal of the diaphysis. A small amount occasionally was distributed to the periosteum.

Each rabbit received from 0.2 to 0.6 cubic centimeter of the agglutinated living or killed suspension and died or was killed from 12 hours to 48 days after operation. A few were injected with non-agglutinated living staphylococci but all died within 2 days. The animals that received killed organisms be-

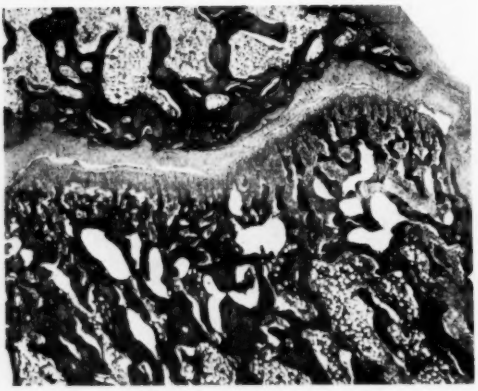


Fig. 10. Acute metaphyseal subchondral necrosis and partial separation of the ossifying portion of the head epiphyseal plate in a femur 14 hours after injection of a partially agglutinated suspension of living cocci. ×16.

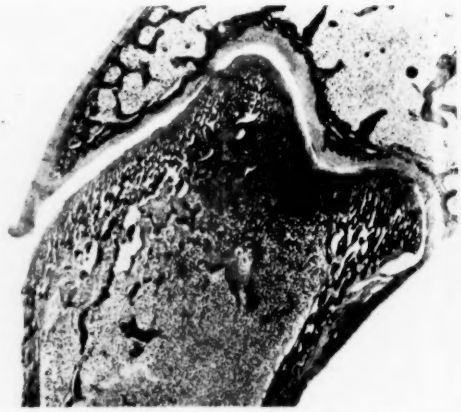


Fig. 11. The distal metaphysis of the femur of Figure 10. Necrosis has extended through to the joint space anteriorly but not posteriorly where periosteal blood supply is more abundant. ×6.5.

haved like normal animals, except occasionally one favored the injected leg in hopping and held it partially flexed at the knee and hip. Several of the animals injected with living organisms died within 24 hours after operation; others lived as long as 17 days, depending upon the mass injected and the viability of the cocci, as determined by culture at the time of operation. The local disability of the leg produced by septic emboli was proportionate to the severity of the osteomyelitis and it varied from slight decreased weight-bearing to marked rigid flexion at the hip and knee. A pure growth of staphylococcus, similar to the strain injected, was obtained from joints with exudate that were contiguous with the femurs injected.

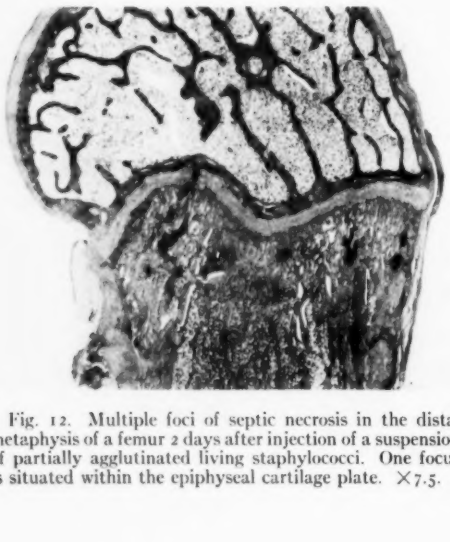


Fig. 12. Multiple foci of septic necrosis in the distal metaphysis of a femur 2 days after injection of a suspension of partially agglutinated living staphylococci. One focus is situated within the epiphyseal cartilage plate. ×7.5.

The femurs were fixed in Klotz solution No. 1 containing formalin to 10 per cent, decalcified with 4 per cent nitric acid and cut into proximal, middle, and distal segments. These blocks were then doubly embedded in celloidin and paraffin from which sections were cut and stained with hæmatoxylin and eosin.

As a further control on anæmic infarction in the femur in young rabbits and for comparison with embolism produced by this strain of agglutinated staphylococcus, one group of animals received a suspension of charcoal in 5 per cent gum acacia according to the above procedure.

RESULTS OF EXPERIMENTS

Agglutinated suspensions of killed Staphylococcus aureus. Gross examination of injected femurs demonstrated infarction and subsequent changes similar in general to those observed after occlusion of the medullary blood vessels by charcoal and significant interference with growth of the bone, ability to bear weight, and of osteogenic reaction within the periosteum. The disturbance in overall length growth of the bone amounted to as much as 2.5 millimeters in 8 days and

Fig. 13. Complete necrosis of the head and its epiphyseal line and focal necrosis of the neck of a femur 8 days after septic infarction by agglutinated living staphylococci. The short neck is depressed and forms a right angle with the shaft. ×5.

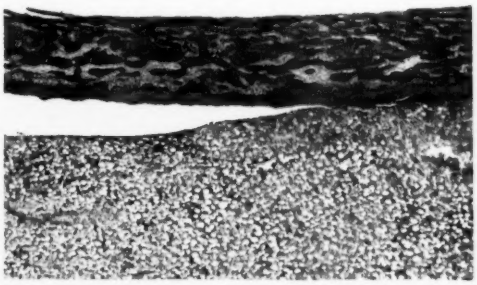


Fig. 14. Septic necrosis of the inner and tissue reaction within the outer lamellæ of the cortex in the femur of Figure 13. There is also partial necrosis in the adjacent marrow tissues. ×16.

this shortening was greatest at the end of the bone receiving the largest amount of the material injected. Femurs infarcted for 3 weeks or more tended to regain this decrease in length. Necrosis in the neck of the femur often produced shortening and medial bending of this portion and allowed the head to droop (coxa vara) and rotate forward. When the head epiphysis was infarcted along with the proximal metaphysis of the shaft it seemed to flatten slightly and overhang the neck at its shelving edge. Occasionally the external surface of the cortex, most prominent near one end of the shaft, was roughened by new bone and the soft parts were removed with some difficulty. Necrosis was not observed in the knee or hip joint capsules or in the other soft tissues of the thigh after injection of killed agglutinated organisms.

Histological preparations of femurs that received killed agglutinated organisms through the nutrient artery to the shaft demonstrated infarcts and anæmic necrosis confined almost entirely to the metaphyses and endosteum. The smaller and more recent infarcts were wedge-shaped and were situated in the tissues immediately beneath the columns of epiphyseal cartilage without noteworthy changes in the medullary tissues of the diaphysis or of the adjacent bony epiphysis. About one-fifth of the epiphyseal cartilage plate, along the edge in apposition with the base of the infarct of the metaphysis, also was necrotic and a definite line of demarcation was present between it and the remaining living portion. Occasionally there was partial separation within the epiphyseal plate along this line. Large infarcts occupied the entire metaphysis and in these the necrosis was most marked about the columns of ossifying cartilage toward the center of the growth region. The infarcts were poorly stained fragmented medullary tissues and devitalized bone and cartilage with considerable peripheral hæmorrhage. When the changes occurred in the cortical bone opposite the metaphysis, there usually were similar regions along the endosteum of the shaft elsewhere but still without noteworthy alteration in the medullary tissues of the central portion of the shaft.

Infarcts present for 6 days or longer demonstrated a separation and reorganization process, present on all sides of the wedge except at the base beneath the epiphyseal cartilage plate. This consisted of many small blood vessels in a loose, poorly cellular and œdematous fibrillar stroma that invaded the débris of the necrotic region. It later became more dense, fibrous with a few clear spaces and a variable number of osteoclastic giant cells. Compact and spongy new bone was formed when the infarct extended to the cortex and often new osseous tissue was laid down outside the cortex in the form of an involucrum.

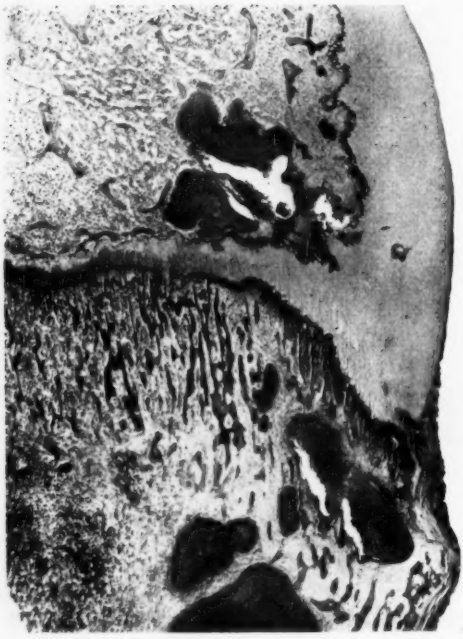


Fig. 15. Multiple subchondral abscesses in the distal metaphysis and epiphysis of a femur 17 days after septic infarction. (Six to eight colonies grew on agar plates inoculated with the suspension at the time of injection.) Foci in this bony epiphysis were unusual. ×16.

The metaphyseal surface of the cartilage plate remained necrotic until organization of the infarct, but the columns above continued to grow without being ossified, so that frequently the growth line was as much as three times its normal width. Overgrowth of the cartilage did not occur at the periphery of the epiphyseal plates because of periosteal and capsular collateral circulation. This was especially true at the posterior edge of the distal plate where many muscle tendons insert and at the lip of the head epiphysis where the capsule of the hip joint is attached to the neck of the femur. As organizing tissues replaced the infarcted region and where the blood supply was adequate, the long columns of epiphyseal cartilage cells narrowed to about their former length and osteoid tissue and new bone were formed in irregularly arranged interlacing spicules. The level of the metaphyseal surface of the plate at the time of

Fig. 16. Marked deformity, scarring, and new bone formation in the proximal end of the femur of Figure 15. There is a distinct line at the level of arrested growth. ×7.5.

infarction was frequently marked by transverse spicules, some of which extended entirely across the marrow cavity. Vessels occasionally pierced the central portion of the growth line from the epiphyseal body and aided in restoring function to the growth center. When the injection of emboli was made such that a bony epiphysis was infarcted with its corresponding metaphysis, there was complete necrosis of the conjugal epiphyseal cartilage and a tendency for it to be narrower than usual and collapsed. This was most marked in the epiphyseal cartilage line of the head of the femur where the force of weight-bearing caused the head to droop and to overhang the neck. Impaired nutrition and growth produced shortage of the neck and its angle with the shaft was more acute than normal.

The primary site of embolic infarction of the femoral epiphyses was beneath the articular or other cartilage and was similar to metaphyseal infarcts beneath epiphyseal lines of the diaphysis. Such infarcts of the head of the femur were usually accompanied by regions of necrosis in the corresponding acetabula and haversian fat tissues at the base of the ligamentum teres.

Necrosis in the femurs produced by carbon emboli was more marked and extensive in the medullary tissues of the shaft than after the injection of clumps of dead cocci. There were

less definite deposits of the material beneath the columns of ossifying epiphyseal cartilage and more occlusion of larger vessels due to the greater size of the carbon particles. Revascularization and organization of the charcoal infarcted tissues required a longer time and the ultimate fibrous tissue replacement was more dense. The killed organisms produced more profound changes in the growth centers and seemed to elicit greater necrotic bone destruction by giant cells (osteoclasts) than the charcoal emboli.

Agglutinated suspensions of living Staphylococcus aureus. The gross alteration in size and contour of femurs infarcted with living were similar to but more marked than in those that received injections of killed organisms. In addition, several joints contiguous to infarcted metaphyses contained exudate from which a pure culture of the organism was obtained. Some of these joints at the distal end of the femur were associated with liquefaction necrosis of the metaphysis and pathological fracture through the epiphyseal cartilage plate. There were no abscesses of the soft tissues.

Microscopic examination demonstrated anæmic necrosis, according to the pattern described for bland infarction, combined with exudative inflammation. The latter agent intensified the hæmorrhages and necrosis and elicited a marked infiltration of polynuclear leucocytes about the septic infarct. This acute pyogenic osteomyelitis very definitely and constantly began in the so called growth regions with a few small foci in the inner layers of the cortex. In preparations of recent infarcts there were masses of cocci wedged to the apices of the lacunar spaces between strands of calcified cartilage matrix. These collections were present also beneath articular and other cartilage of infarcted epiphyses and occasionally within the haversian canals of the cortex, but not in the medullary tissues of the shaft. The intimate association of the site of beginning abscess formation with the very tips of the spaces between ossifying strands of calcified cartilage matrix was striking. Partial or complete separation regularly occurred within the epiphyseal cartilage plate at the base of the metaphyseal infarcts. The line of separation in histological preparations was straight and uniform. Its position corresponded to the limits of cartilage necrosis and to the line demarcating the extent of necrosis in epiphyseal plates at the base of infarcts produced by bland bacterial emboli. That portion (about 80 per cent of the width of the plate) remaining attached to the bony epiphysis usually maintained its viability except when the epiphysis was infarcted with the metaphysis. There often was a narrow localized zone of tissue reaction on the epiphyseal side of the cartilage plate after septic infarction of the growth center but this was slight compared to the reaction on the metaphyseal side of the conjugal cartilage.

When the metaphyseal abscesses were sufficiently large to extend to the cortex, there was exudate in the canaliculi of the bone and occasionally beneath the periosteum. Necrosis of the subperiosteal tissues elicited a marked cellular and fibrous tissue reaction of the periosteum, dead bone absorption, and new bone formation. Complete separation of the epiphysis occurred when the walling-off process here at the periphery of the cartilage plate was unable to maintain viable tissues between epiphysis and diaphysis. The amount of new bone formation (involucrum) about the infarcted portion of the shaft depended upon the amount of damage to the cortex and it varied from none to a wide layer. Occasionally, the periosteum also was necrotic, in which event no involucrum formed and consequently the metaphyseal portion of the shaft collapsed.

The walling-off and reorganization process in septic infarcts seemed to proceed more slowly than those produced by bland staphylococcus emboli. Septic necrosis produced more new cortical and spongy bone, greater injury to the epiphyseal plates, and more disarrangement of the medullary tissues of the metaphyses than did the aseptic infarcts. In a few bones there were circumscribed regions of necrosis like encapsulated abscesses and with the return of growth activity in the corresponding cartilage line, these were pushed farther away from the epiphyses. The new bone formed outside infarcts of the endosteal

tissues was always well delimited from the dead cortical bone by cellular fibrous tissues containing osteoclastic giant cells, but sequestration was not observed.

EVALUATION OF EXPERIMENTS

The intra-arterial injection of acid killed and agglutinated staphylococci through nutrient vessels of the femur in growing rabbits has demonstrated that infarcts and subsequent changes of bone can be produced by bland bacterial emboli. These infarcts resemble those produced by a particulate suspension of charcoal and probably no chemical factor from disintegrating bacteria is added to the embolic disturbance of circulation. The clumps of cocci were considerably smaller than the individual carbon particles and fewer particulate bacterial masses were injected. For this reason, and because the bacteria were more readily removed from the tissues than the carbon, the infarcts were smaller; they stimulated less tissue reaction and healed more rapidly. Where the emboli contained living bacteria certain changes were noted which relate to septic and aseptic diseases of bone.

The wedge-shaped regions of necrosis were intimately associated with so called growth centers because of the peculiar vascular arrangement and blood supply of subchondral tissues. The localization of the infarcts and collections of living staphylococci between the strands of calcified epiphyseal cartilage demonstrate that this is the site of lodgement of embolic material. Killed organisms were not demonstrated directly beneath the ossifying cartilage because the bacteria were small in number and rapidly disintegrated in the tissues. Carbon particles did not reach these tissues as a rule because they were too large for the caliber of the vessels. The terminal capillaries in the finger-like tissue spaces of the metaphyses, which erode the epiphyseal cartilage matrix in the process of growth, probably are not loops but rather are thin-walled tubes which elongate as the erosion of cartilage progresses. The absence of abscesses in the soft tissues, even when living agglutinated bacteria were injected into the deep femoral artery, supports this belief. Apparently the clumps were sufficiently small to pass through the usual capillary bed. Necrosis beneath ossifying cartilage and epiphyseal separation, even when the cocci were not agglutinated, also favor a distinctive capillary pattern in the growth centers. This capillary arrangement is present beneath all cartilage of the epiphyses, but the number of actively proliferating vessels is probably dependent upon the amount of bone formation. Since growth in length occurs in the metaphyses, these tissues are more vulnerable than the subchondral tissues of the epiphyses where growth is by expansion. Following embolic occlusion of the capillaries there was retrograde thrombosis, the extent of which depended upon the size and number of particles injected and the collateral circulation. The tissue changes with infection added to this vascular disturbance by mycotic emboli depended upon the virulence and mass of living organisms. Therefore, it is possible, clinically, to have all stages between anæmic infarcts and acute abscesses from bacterial emboli, either pyogenic or specific as tuberculosis.

The tissues in the center of the metaphyses were more vulnerable than those beneath the periphery of the epiphyseal cartilage plates because the latter have a periosteal blood supply. This was demonstrated by regular occurrence of small infarcts beneath the center of the epiphyseal cartilage and by ingrowth of blood vessels and reaction tissues from the peripheral portions of the metaphysis and from the periosteum. Further, necrosis of the metaphyseal ends of the epiphyseal cartilage columns and interference with ossification were not so marked near the cortex, particularly in regions opposite the insertion of muscles and tendons. Infarcts at the distal end of the femur tended to be slightly above the center of the plate since the articular cartilage extends far up the bone anteriorly and the periosteum has no muscle attachments. Complete separation in the epiphyseal line occurred only when the region of necrosis included the cortical and periosteal tissues of the metaphysis.

Assuming that periosteal collateral circulation to metaphyses is a constant or uniform

potential source of nutrition, the vulnerability of a given growth center is proportionate to its size. For this reason metaphyseal infarcts, septic or aseptic, more often occur in large bones, such as the femur, tibia, and humerus, and in these subjacent to the broad epiphyseal cartilage plates. Also, other things being equal, infarcts occur most commonly where there is greatest growth activity, since it is here that compact strands of calcified matrix and the lacunar spaces between them are longest and have many developing capillaries that project into the eroded cartilage. Subchondral foci of infection and bland infarcts may occur in the epiphyses after the epiphyseal lines are completely ossified so long as there is enchondral ossification in the secondary centers. The predominance of lesions in some over other small bones probably concerns individual differences in structure and blood supply. External trauma may localize an infection but only in so far as it disturbs the vascular pattern of a portion of bone or accentuates the dearth of collateral circulation in regions that are normally more susceptible to infection and nutritional disturbances.

The uniform occurrence of necrosis at the base of the infarcts in the metaphyseal ends of the columns of epiphyseal cartilage demonstrates that these tissues which are about to be converted into spongy bone and osteoid tissue have a diaphyseal blood supply and that there is no noteworthy vascular communication between the epiphyses and diaphysis. The amount of the necrosis in the cartilage plate was constant and marked the level where complete separation within the line occurred when the necrosis extended laterally to include the cortex and periosteum. Since the columns of cartilage continued to grow from above, even after complete separation within the line, cartilage proliferation is dependent upon epiphyseal blood supply and should be distinguished from ossification. Necrosis of the plate occurred only when its bony epiphysis was infarcted. A collateral circulation to the metaphyses from the epiphyses through this conjugal cartilage was formed in some of the infarcted metaphyses. Occasionally, after marked infarction of the metaphysis, the distal end of the femur had a club-shaped deformity due to the relative expansile overgrowth of the knee epiphysis.

Bland infarcts and abscesses localized also beneath the endosteum and within cortical bone. This suggests that the capillary arrangement among spicules of spongy bone and within haversian system to some degree may be similar to the vascular pattern described for enchondral ossifying tissues. However, the extent of involvement of the cortex opposite an infarct of the metaphysis was not constant as if limited, and it seemed to be dependent upon the size of the infarct. Septic infarcts extended along the epiphyseal cartilage plate and included considerably more compact bone than those produced by a similar injection of killed organisms. The bacteria from septic foci of necrosis penetrated the subperiosteal tissues and produced suppurative arthritis. The retrograde extension of osteomyelitis from the metaphysis to the more central portions of the shaft was considerably less than its spread laterally due to the abundant blood supply of the central medullary tissues. Some of the abscesses of longer duration were farther from the epiphyseal lines because of reorganization and return of function to the growth centers. Involucrum formation seemed to be stimulated by the presence of necrotic compact bone and was present only where the necrosis had not included the periosteum. This was similar to new bone formation by the periosteum observed after infarction of the femur with charcoal emboli (18). The removal of dead bone by osteoclastic giant cells seemed considerably more active in infarcts produced by staphylococci, living or dead, than in those caused by charcoal emboli.

Deformity results when a portion of a bone is unable to withstand the stresses placed upon it. The alteration in contour depends upon the amount of injury to normal structure present within the bone and to the forces which tend to produce distortion. The type and virulence of bacteria, when present in foci of necroses, are important in determining the amount and acuteness of the disease and subsequent deformity. Bacterial infarction of the proximal end of the femur produced various degrees of collapse and hypoplasia of the

neck (coxa vara) and the neck rotated anteriorly so that the entire femur was displaced inward slightly. Infarcts in the head of the femur caused this epiphysis to flatten and overhang the neck at its shelving edge. Femurs infarcted for 3 weeks or more partially regained the growth deficiency either by hyperplasia in an unaffected growth center or organization and resumption of function of the infarcted metaphysis.

Transverse spicules of bone often were observed in infarcted metaphyses after organization and return of function to the growth centers. These marked the level of the ends of epiphyseal cartilage columns at the time of infarction and they correspond to the so-called lines of arrested growth described by many authors (6, 14, 25) in various acute illnesses and metabolic disturbances.

SUMMARY

Masses of bacteria, living or dead, may act as emboli in producing septic or aseptic infarcts in bone. The subsequent changes are modified by infection when the infarcts are produced by infected emboli.

These infarcts occur regularly beneath ossifying cartilage, especially in the metaphyses, due to relative poor collateral circulation in subchondral tissues and to the presence of open end-capillaries immediately beneath ossifying columns of cartilage. The occurrence of foci in some metaphyses more than in others probably is dependent upon the diameter of the epiphyseal cartilage plate and the rate of bone growth within the metaphysis.

The extent of bone destruction and subsequent distortion depends upon size of the infarct, and if infected, further upon the mass and virulence of the bacteria. Exudative arthritis may follow the spread of a metaphyseal focus of infection along the epiphyseal line to the periosteum.

Metaphyseal growth centers and the adjacent portion of epiphyseal cartilages obtain their blood supply from the periosteum and nutrient vessels of the shaft. The epiphyseal portion of the cartilage plates, about four-fifths of the thickness, depends upon the circulation of the epiphysis. Separation of epiphysis and diaphysis occurs along a plane between the limits of the two sources of nutrition within the conjugal cartilage plate.

The stimulus of necrotic cortical bone and adequate periosteal blood supply are necessary for the formation of new compact bone (involucrum).

Lines of arrested growth may be present in metaphyseal infarcts produced by clumps of living or dead staphylococcus aureus.

BIBLIOGRAPHY

1. Axhausen, G. Ueber anaemische infarkte am Knochensystem und ihre Bedeutung fuer die Lehre von den primaeren Epiphyseonekrosen. Arch. f. klin. Chir., 1928, 151: 72.
2. Idem. Knochennekrose und Sequesterbildung. Deutsche med. Wchnschr., 1914, 40: 1.
3. Bohlman, H. R. Experiments with foreign materials in the region of the epiphyseal cartilage plate of growing bones to increase their longitudinal growth. J. Bone & Joint Surg., 1929, 11: 365.
4. Compère, E. L. Streptococcus viridans osteomyelitis. J. Bone & Joint Surg., 1932, 14: 244.
5. Eliason, E. L., and Ferguson, L. K. Epiphyseal separation of the long bones. Surg., Gynec. & Obst., 1934, 58: 85.
6. Eliot, M. M., Souther, S. P., and Park, E. A. Transverse lines in X-ray plates of the long bones of children. Johns Hopkins Hosp. Bull., 1927, 41: 364.
7. Greenwood, H. H. Relation of tuberculosis to Koehler's disease. Brit. J. Surg., 1927, 15: 245.
8. Haas, S. L. The relation of the blood supply to the longitudinal growth of bone. Am. J. Orthop. Surg., 1917, 15: 157.
9. Idem. The localization of the growing point in the epiphyseal cartilage plate of bones. Am. J. Orthop. Surg., 1917, 15: 563.
10. Haldeman, K. O. Acute osteomyelitis. Surg., Gynec. & Obst., 1934, 59: 25.
11. Hallermann, W. Ueber die Beziehungen zwischen Osteoporose und arteriosklerotischen Veraenderungen der ernaehrenden Knochengefaesse am oberen Femurende. Arch. f. klin. Chir., 1928, 150: 683.
12. Harbin, M., and Zollinger, R. Osteochondritis of the growth centers. Surg., Gynec. & Obst., 1930, 51: 145.
13. Harris, H. A. The vascular supply of bone, with special reference to the epiphyseal cartilage. J. Anat., 1929, 64: 3.
14. Idem. Lines of arrested growth in the long bones in childhood. The correlation of histological and radiographic appearances in clinical and experimental conditions. Brit. J. Radiol., 1931, 4: 561.
15. Jackson, L. Experimental streptococcal arthritis in rabbits. J. Infect. Dis., 1913, 12: 364.
16. Jahn, P. Beitraege zur Kenntnis der histologischen Vorgaenge bei der Wachstumsbehinderung der Roehrenknochen durch Verletzungen des Intermediarknorpel, Morphologische Arbeiten von. Schwalbe. 1892, 1: 241. (Quoted from Haas.)
17. Kistler, G. H. Sequences of experimental infarction of the femur in rabbits. Arch. Surg., 1934, 29: 589.

18. Idem. Formation of bone by the periosteum after experimental infarction by embolism of femur in rabbits. Proc. Soc. Exper. Biol. & Med., 1934, 31: 1218.
19. KOLODNY, A. The architecture and blood supply of the head and neck of the femur and their importance in the pathology of fractures of the neck. J. Bone & Joint Surg., 1925, 7: 575.
20. KOENIG, F. Ueber freie Koerper in den Gelenken. Deutsche Ztschr. f. Chir., 1887, 27: 90.
21. KUWAHATA, H. Neue experimentelle Untersuchungen ueber die Entstehung der akuten eitrigen Osteomyelitis. Deutsche Ztschr. f. Chir., 1930, 222: 374.
22. LEXER, E. Die Entstehung entzuendlicher Knochenherde und ihre Beziehung zu den Arterienversweigungen der Knochen. Arch. f. klin. Chir., 1903, 71: 1.
23. LEXER, E., and KULIGA, P. Untersuchungen ueber Knochenarterien Mittelst Roentgenaufnahmen injizierter Knochen und ihre Bedeutung fuer einzelne pathologische Vorgaenge am Knochensysteme. Vol. 8. Berlin: A. Hirschwald, 1904.
24. MEISENBACH, R. O. A consideration of the chemical and mechanical stimulation of bone with reference to the epiphyseal and diaphyseal lines: results of animal experimentation. Am. J. Orthop. Surg., 1910, 8: 28.
25. MILLER, L. F., and RUBELL, I. Transverse bands in the bones of a tuberculous child. Am. J. Dis. Child., 1934, 47: 354.
26. MUELLER, W. Deutsche Ztschr. f. Chir., 1887, Vol. 25.
27. OBERZIMMER, J. Formazione di necrosi circoscritte e di sequestri nella tubercolosi ossea. Chir. d. organi di movimento, 1933, 18: 317.
28. PEASE, C. N. The relation of streptococcus viridans to apophysitis of the tibial tubercle. Am. J. Surg., 1934, 24: 149.
29. PHEMISTER, D. B. Newer knowledge of pyogenic infections of bone. Proc. Inst. Med. Chicago, 1929, 7: 169.
30. Idem. Repair of bone in presence of aseptic necrosis resulting from fractures, transplantations, and vascular obstruction. J. Bone & Joint Surg., 1930, 12: 769.
31. Idem. Silent foci of localized osteomyelitis. J. Am. M. Ass., 1924, 82: 1311.
32. Idem. Chronic fibrous osteomyelitis. Ann. Surg., 1929, 90: 756.
33. PHEMISTER, D. B., BRUNSCHWIG, A., and DAY, L. Streptococcus infections of the epiphyses and short bones, and their relation to Legg-Perthes' disease, Kienboeck's disease and Koehler's disease of the tarsal navicular. J. Am. M. Ass., 1930, 95: 995.
34. REISCHAUER, F. Trauma und haematogene Knocheninfektion. Beitr. z. klin. Chir., 1932, 156: 411.
35. SCHULZE, W. Ueber die Ursachen der Bakterienablagerung im Knochen. Arch. f. klin. Chir., 1933, 177: 450.
36. SENN, N. Principles of Surgery. 4th ed., p. 278. Philadelphia: F. A. Davis Co., 1909.
37. SEVER, J. W. Changes in the epiphysis—secondary to infection. New England J. Med., 1933, 209: 62.
38. STARR, C. L. Acute hematogenous osteomyelitis. Arch. Surg., 1922, 4: 567.
39. VOGT, P. Die traumatische Epiphysentrennungun, deren Einfluss auf das Laengenwachstum der Roehrenknochen. Arch. f. klin. Chir., 1878, 22: 343.
40. WAKELEY, C. P. G. Acute osteomyelitis in children. Brit. M. J., 1932, 2: 752.
41. WARDLE, E. N. The etiology and treatment of slipped epiphysis of the head of the femur. Brit. J. Surg., 1933, 21: 313.
42. WILENSKY, A. O. The mechanism of acute bacterial infection of a joint. Arch. Surg., 1926, 13: 895.

SYMPATHETECTOMY AS A PRELIMINARY TO THE OBLITERATION OF POPLITEAL ANEURISMS

With a Suggestion as to Sympathetic Block in Cases of Ligature, Suture, or Thrombosis of Large Arteries[1]

CLARENCE E. BIRD, M.D., F.A.C.S., Louisville, Kentucky

THE incidence of extensive gangrene requiring amputation following operations for the relief of popliteal aneurisms is estimated by the writer to rest between 8 and 15 per cent. This approximation takes into consideration a probable number of unsuccessful cases which are not reported in the literature, and for this reason the figure is higher than that obtained by Professor Matas (6) who found among 154 personal and reported cases of popliteal aneurism operated upon by his three methods of aneurismorrhaphy up to 1915, 8 cases of gangrene, or 5.2 per cent.

Doctor Matas' personal success has been extraordinary and the papers indicate that in his own cases he has had practically no gangrene. This splendid achievement has been obtained, first, by the use of his compressor (4) and tests for determining the adequacy of the collateral circulation (5), second, by a correct clinical judgment as to the type of aneurismorrhaphy indicated, and, finally, by strict adherence to the principles of his operations (3).

Nevertheless, if others share my feeling, they approach the obliteration of a popliteal aneurism with misgivings, and will be grateful for any aid which may provide further assurance that the collateral circulation will be adequate. Twice in cases of my own I have observed the development of a false aneurism during the preparatory 2 or 3 weeks of repeated compression of the femoral artery above the sac by Matas' method. In neither instance was any direct pressure exerted upon the sac itself, and I do not believe that the false aneurisms arose because of instrumental damage to the walls of the true sacs. The presence of an enlarging false aneurism causes compression of the motor and sensory nerve fibers, increases the discomfort of the patient, hurries the operator to carry out his procedure before the time he would otherwise elect, and increases the likelihood of infection in the field, either before or after the operation, or both. An advantage would be gained if an obliteration could be done safely at any time after admission to the hospital, even almost immediately, if indicated.

In the first case of which I speak, the collateral circulation as tested by the method of Matas was doubtfully satisfactory. The dorsalis pedis and posterior tibial arteries were palpable but were weaker on the side of the aneurism. A sympathetectomy was considered but was not carried out. Immediately after the obliteration of the sac the appearance of the toes and foot was discouraging, and the outcome, in brief, was a mid thigh amputation necessitated by gangrene.

In order to avoid a possible repetition of this unfavorable result it was determined, in the second patient, to take advantage of the work of Mulvihill and Harvey, who showed by well controlled experiments on dogs the tremendous advantage to be gained in the immediate postoperative period if a sympathetectomy is done at the time of the ligation of the external iliac artery. When the sympathetectomy was not carried out, the subcutaneous temperature of the corresponding foot dropped gradually over a period of 2 to 7 hours to, or almost to, room temperature, remained there for from 2 to 6 hours, then gradually rose to body temperature on an average of 13 hours after the ligation of the external iliac artery. On the other hand, when the sympathetectomy had been performed the drop in temperature did not occur. Sympathetectomy prevented, therefore, an 8 to 14 hour period of partial ischæmia. It is assumed that these experiments on dogs may be applied directly to the popliteal artery of man, and there appears to be no reason to doubt the correctness of this view. Such ischæmia, if applied to an already diseased

[1]From the Department of Surgery of the University of Louisville School of Medicine and the Surgical Service of the Louisville City Hospital.

human leg, could hardly fail to be damaging or disastrous. Sympathetectomy, on the other hand, should provide a wide margin of safety.

A short summary of the pertinent findings in the record of the second patient follows:

W. R., (L. C. H. No. 35926), a single, colored dishwasher, aged 29 years, was admitted on June 1, 1934. He first noted a swelling in the right popliteal space 2 months before. This rapidly enlarged causing him to limp because of inability to extend the knee joint completely. Pain, extending down the leg, began 2 weeks before admission. He had had a penile sore several years previously but had not been treated by inunctions or injections. There was no history of trauma to the popliteal space. The patient had been troubled "all his life" by frequent slight nausea, and copious, effortless regurgitation of food. These symptoms had not been relieved by the removal of his appendix 10 years previously.

Examination showed normal development and slight undernourishment. The pupils and eyegrounds were normal. There was no perforation of the nasal septum. The teeth were carious, the gums infected. Fluoroscopic examination and barium studies revealed a thoracic stomach which extended from the upper aperture of the thorax to the level of the diaphragm, where, after some delay, the barium emptied directly into the small bowel. The balance of the intestinal tract appeared normal. The heart was slightly enlarged and an electrocardiogram showed borderline right axis deviation and sinus arrhythmia. The radial pulses were equal, strong, regular, without sclerosis; rate 88. Blood pressure registered 120/80. There was an old lower right rectus operative scar. The knee jerks were present and approximately equal on the two sides.

In the right popliteal space was a large, tense, expansilely pulsating tumor over which, in certain positions of the limb, a delicate thrill could be felt. A loud bruit with systolic accentuation was heard over the entire tumor. The skin and deep fascia were tense over the mass, the borders of which could not be made out. The knee, leg and foot were moderately swollen and the veins below the level of the tumor were engorged. Elevation only partly emptied them. There was moderate pitting œdema of the extremity on the side of the aneurism. The feet and legs were moist and equally warm to touch. Flexion and extension of the knee were limited by painful tension in the region of the mass and by pain shooting down the leg. The dorsalis pedis and posterior tibial arteries were unusually easy to palpate on the left, but on the right no pulsations could be felt. The dorsalis pedis artery on the right stood out as a pulseless cord. The femoral pulses were normal and obliteration of the right femoral artery by pressure did not change the heart-rate. Obliteration of this vessel stopped the expansile pulsation, thrill, and bruit in the popliteal mass and on elevation of the leg during compression the mass became soft and plastic. The appearance of the knee suggested fluid in the joint but the patella did not float. There were no gross sensory changes in either leg, but there was weakness of the anterior tibial and peroneal groups of muscles on the right. Injection of neoskiodan into the right femoral artery produced an imperfect visualization of an almost spherical mass 6 centimeters in diameter in the midpopliteal region. No collaterals were seen.

The Matas test, carried out with a mechanical constrictor and an Esmarch bandage, showed return of blood to the subungual capillaries in from 2 to 5 minutes after removal of the bandage. Observation of the descending flush, however, was difficult to make in the presence of the black skin, and further, we were unable to be absolutely sure that some blood was not occasionally leaking past the constrictor. The veins did not fill well from below and my impression was, on the whole, that we were dealing with an unsatisfactory collateral circulation.

The blood Wassermann reaction was four plus; red blood cells, 4,050,000; hæmoglobin (T), 80 per cent; white blood cells, 7,750; differential count and smear normal. The urine showed one plus albumin and an occasional pus cell. Analysis of the fluid obtained from the thoracic stomach by a nasal tube with its tip 32 centimeters from the nostril showed a specific gravity of 1.032, free hydrochloric acid 28, and total acid 46.

The temperature varied from subnormal to 100 degrees F., with a pulse of 80 to 100. Compression of the femoral artery just above the adductor hiatus was carried out for 5 to 10 minutes every 2 hours each day. Shortly the patient began to complain of increased pain in the knee and leg, and the tumor and all of the tissues of the knee and leg enlarged. The knee was held flexed to 100 degrees for comfort.

On June 9, 1934, a right lumbar sympathetectomy was performed under spinal anæsthesia, severing the rami and removing the ganglia and trunk between the levels of the second lumbar and the first sacral vertebræ. A warm, dry leg resulted, which, in contrast to the left, was hyperæmic and exhibited a diffuse, dusky red blush throughout. The slight fever persisted and the pain became more constant as the popliteal swelling increased in tenseness. The Matas test was now more difficult to carry out on account of the pain and because of the necessity of flexion, but there was no longer any doubt as to the adequacy of the collateral circulation. After removal of the Esmarch bandage there was always a return of color to the sole of the foot and to the tissues beneath the nails within one minute and a half. No pulsation returned to the dorsalis pedis and posterior tibial arteries. The weakness of the anterior tibial and peroneal muscles became a total paralysis with marked hypæsthesia of the deep and superficial peroneal areas.

On July 5, 1934, an obliterative endoaneurismorrhaphy was carried out under spinal anæsthesia. A

curved, longitudinal incision, convex medially, was made over the popliteal space. The semimembranous muscle was found thinned out and displaced. No nerve trunks, large veins, or lymph nodes were exposed. The deep fascia was incised over about 8 centimeters of the aneurismal surface, which was discolored with blood pigment and somewhat œdematous. The circulation was temporarily occluded by a blood pressure cuff which had been placed around the upper thigh. The sac when opened longitudinally was found to be 3 millimeters thick, and contained fresh and old blood clots as large as one and one-half adult fists. The posterior surfaces of the femur and tibia were palpated but there was no break through to the bone or joint space. The endothelium was intact about the upper and lower orifices and along the external side of the sac, but elsewhere the lining was shaggy and false and extended upward and downward irregularly between the muscular layers. Closure of the upper and lower orifices was accomplished with fine interrupted silk sutures. On removal of the pressure from the femoral artery above, there was only slight oozing. One geniculate vessel was closed from the inside and the large sac was obliterated with three layers of interrupted silk sutures. The deep and superficial fascia was closed with interrupted silk without drainage. A loose bandage and posterior plaster splint were applied and the patient was returned to bed with the leg slightly below the level of the heart and with a warm cradle over the extremities.

From the first there was no doubt as to the adequacy of the circulation. Color returned to the nails almost immediately after release of compression and the return was as rapid or slightly more rapid than on the opposite side. Forty-eight hours after operation there could be felt and seen a pulsating artery beneath the skin on the anteromedial surface of the knee. The dorsalis pedis and posterior tibial vessels on the right remained pulseless. The slight fever and the peroneal paralysis persisted and the deep tissues beneath the cleanly healed external wound gradually became more swollen. On the nineteenth day after operation a soft, deep, indolent abscess was opened, and about 200 cubic centimeters of odorless, Staphylococcus aureus pus was evacuated. Thereupon the temperature returned to a consistent normal for the first time since admission and the wound healed rapidly. The motor paralysis was still present on discharge on July 31, 1934.

Had it not been that, due to the enlarging false aneurism and the pain, the sac must be obliterated promptly, I should have been tempted to await the arrival of the pavaex apparatus (2) which Doctors Mont Reid and Louis Herrmann have so kindly sent us from Cincinnati for use in the vascular clinic; and I would feel more enthusiasm in reporting the evident benefit of preliminary sympathetectomy in this case, were it not for the fact that the procedure will without doubt shortly be superseded by the use of their effective stimulant of collateral circulation. By its use the diameter of the collateral arteries can not only be increased before operation, but the entire limb may be placed in the boot under negative pressure immediately after operation, in this way counteracting the tendency toward sympathetic overaction in a limb recently traumatized. In fact, the limb will be subjected to the negative pressure before the passive dilatation due to the spinal anæsthetic, if such be used, has worn off, so that the precious collaterals may never be subjected to the action of the sympathetic fibers until the period of jeopardy is passed. It is true, of course, that during the period before this apparatus becomes generally available, the procedure of sympathetectomy, or sympathetic block, may be of use in the cases under consideration, and also in the management of other aneurisms and tumors which involve large arteries of the extremities (1).[1]

A further suggestion may be made. It is probable that a temporary block of the appropriate portions of the sympathetic chain with alcohol will continue to be valuable in those cases in which, because of the presence of fracture apparatus or dressings, the pavaex apparatus cannot be utilized. I have in mind especially serious fractures or injuries because of which it is found necessary to suture or tie large arteries or those cases in which there is present arterial thrombosis which plays a devastating part.

SUMMARY

Lumbar ganglionectomy was performed as a preliminary to the obliteration of a popliteal aneurism, thereby avoiding the constrictor action of the sympathetic fibers on the collateral arteries during the dangerous early postoperative period.

In the same way advantage should be derived from sympathetic ganglionectomy or sympathetic block carried out before or immediately after operations or injuries which

[1] The preliminary sympathetectomy reported is similar to the sympathetic block subsequently found to have been resorted to by Gage in the operative management of a case of mycotic aneurism of the common iliac artery.

involve obstruction or threatened obstruction to large arteries of the extremities.

The rationale for these procedures is contained in the work of Mulvihill and Harvey.

In suitable cases the pavaex apparatus of Hermann and Reid should accomplish the same result without interference with the sympathetic fibers themselves.

REFERENCES

1. SAGE, MIMS. Mycotic aneurysm of the common iliac artery; sympathetic ganglion block as an aid in the development of the collateral circulation in arterial aneurysm of peripheral arteries; report of a case. Am. J. Surg., 1934, 24: 667–710.
2. HERRMANN, L. G., and REID, M. R. Pavaex (passive vascular exercise) treatment of obliterative arterial diseases of extremities. J. Med., 1933, 14: 524–529.
3. MATAS, RUDOLPH. An operation for the radical cure of aneurism based upon arteriorrhaphy. Ann. Surg., 1903, 37: 161–196.
4. Idem. Tests to determine efficiency of collateral circulation before attempting permanent occlusion of great surgical arteries. Ann. Surg., 1910, 52: 126–130.
5. Idem. Testing the efficiency of the collateral circulation as a preliminary to the occlusion of the great surgical arteries. J. Am. M. Ass., 1914, 63: 1441–1447.
6. Idem. Endo-aneurismorrhaphy. Surg., Gynec. & Obst., 1920, 30: 456–459.
7. MULVIHILL, D. A., and HARVEY, S. C. Studies on collateral circulation. I. Thermic changes after arterial ligation and ganglionectomy. J. Clin. Invest., 1931, 10: 423-429.

OBSTRUCTIVE LESIONS OF THE UTERUS AND THEIR COMPLICATIONS[1]

ARTHUR H. CURTIS, M.D., F.A.C.S., CHICAGO, ILLINOIS

A REALIZATION that cervical strictures are of clinical importance was impressed upon me very gradually incidental to interest in the pathology of chronic leucorrhœa. A survey of my records reveals frequent notations of cervical strictures as early as 1920. Yet I did not awaken to an appreciation of their true significance until some years later.

In 1929, at the inaugural meeting of the Baltimore Obstetrical and Gynecological Society, I discussed the incidence and clinical importance of cervical obstructions.[2] To the best of my knowledge, these lesions, other than complete stenosis, and hydrometra and pyometra incident to cancer and to radium therapy, had previously been recognized only as pathological curiosities, and there is apparently still a tendency to overlook their rôle in the etiology of pelvic disturbances.

[2] It is scarcely necessary to emphasize that the impervious cervical canal of many elderly women is almost physiological.

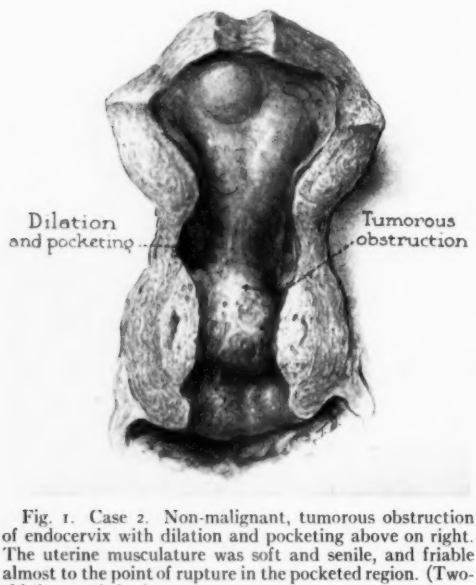


Fig. 1. Case 2. Non-malignant, tumorous obstruction of endocervix with dilation and pocketing above on right. The uterine musculature was soft and senile, and friable almost to the point of rupture in the pocketed region. (Two-thirds actual size.)

The frequency of obstructions of the cervix should again be emphasized; they are very common indeed. But my chief aim at this time is a presentation of important pathological changes which are encountered in the more serious cases, in the hope of arousing greater interest in this subject.

CASE 1. V. B., aged 54 years, with a history of a normal menopause at 48 years, suddenly developed a profuse purulent leucorrhœa 1 month prior to entrance into the hospital. Aside from having given birth to three healthy children after normal labors, the previous obstetrical and gynecological history was negative. There had been no instrumentation or infection to account for the sudden appearance of the purulent discharge.

Attempted dilation under anæsthesia revealed a canal so firmly strictured that the obstruction could not be overcome, and a vaginal hysterectomy was resorted to. The uterus, opened by anterior incision after removal, had a constricted granular canal with multiple firm cicatricial bands and pocketed chambers containing purulent material above the various sites of obstruction.

This case, with multiple strictures and pocketings of the uterus of unknown etiology, complicated by infection of the retained secretion eventuating in postmenopausal profuse purulent leucorrhœa, exemplifies the pathology which is often responsible for "senile vaginitis." With cessation of the menstrual function and shrinkage of the cervical lumen, lesser lesions of varied etiology which cause no annoyance in earlier years suffice to make complete obstruction. There is first a retention of mucoid secretion, then contamination of the retained material, and, finally, a pyometra, or, more commonly a seepage through of the purulent accumulation when pressure sufficient to overcome the obstruction has developed within the uterine cavity. A spontaneous cure may result, but dilation or more extensive surgery is usually required.

CASE 2. E. B., aged 67 years, married 43 years, entered Passavant Memorial Hospital July 23, 1934. She had one child, at the age of 32 years, after a difi-

[1] Presented before the New York Obstetrical Society, January 8, 1935, and before the Society of Surgeons of New Jersey, January 9, 1935. From the Department of Obstetrics and Gynecology, Northwestern University Medical School and Passavant Memorial Hospital, Chicago.

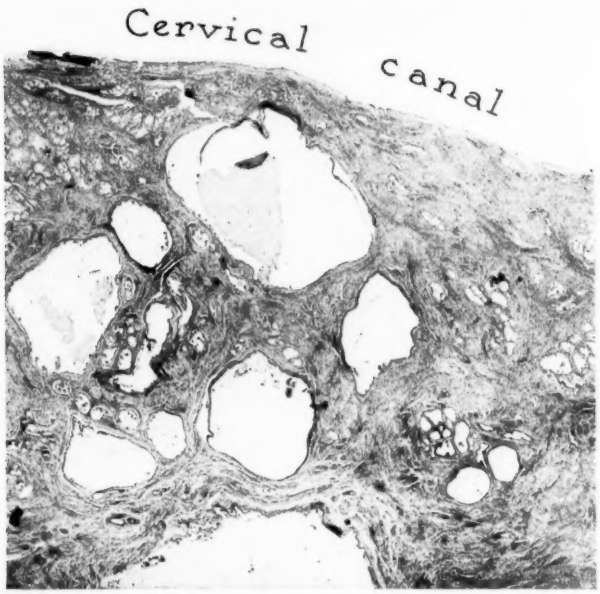


Fig. 2. Case 2. Low power photomicrograph of endocervical tumefaction, consisting of a stroma honeycombed with a multitude of glands and cystic gland spaces. × 9½.

cult spontaneous labor with normal recovery. Menstruation, always normal, had ceased suddenly at the age of 50.

The patient complained of a watery leucorrhœal discharge of many years' duration, sufficient to require a pad. Occasional slight dark bloody spotting had been present for 2 years; a profuse gush of thin dark fluid occurred 2 days before entrance. Examination revealed a large cystocele and a slight amount of dark brown discharge from the uterus, which was of generous size and descended nearly to the vulvar orifice. The cervix was considerably lacerated, the perineum relaxed.

Operation. Dilation, accomplished without difficulty despite an obstruction in the upper cervix, was accompanied by passage of several cubic centimeters of mucoid and watery brown retained secretion. Curettage was abandoned because the uterine wall was found to be soft and thin, and perforation appeared imminent. The prolapsed uterus was then removed, vaginally, normal tubes and ovaries were palpated, the cystocele was corrected and the perineum repaired.

When the soft senile uterus, which was 4 inches in length, was opened, a tumefaction high in the endocervix was bisected. A similar nodulation, in size and appearance resembling the surface of a large preserved strawberry, was present on the posterior wall and terminated above at the level of the internal os of the greatly elongated cervix. The body of the uterus had a capacious cavity, especially in the lower uterine segment, which was pocketed on the right; the uterine wall was soft and senile, thin, and friable almost to the point of rupture in the pocketed region above the tumorous obstruction. Remaining traces of accumulated dark fluid were still present here. In the wall of the fundus were a few small fibroids, one protruding into the cavity (Fig. 1).

Microscopic examination revealed the tumefaction of the endocervix to be a non-malignant growth, consisting of a stroma honeycombed with a multitude of glands and cystic gland spaces of varied size up to many millimeters in diameter. Active pro-

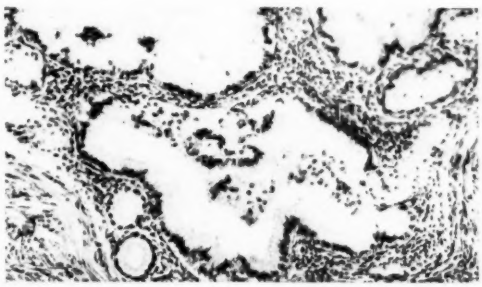


Fig. 3. Case 2. High power photomicrograph of hyperplastic endocervical glands (see Fig. 2). × 125.

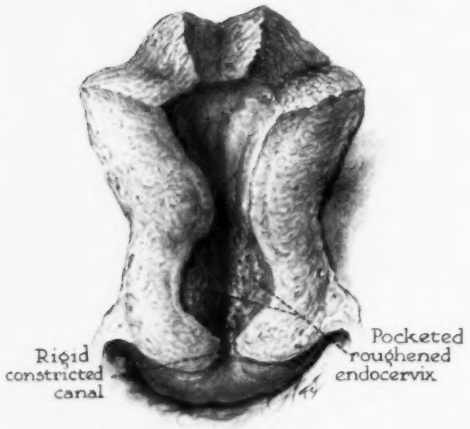


Fig. 4 Case 3. The external os was stomatous; the cervical canal for ¾ centimeter above was narrowed and rigid, passing over suddenly into a sacculated endocervix, the wall of which was roughened and furrowed. (Two-thirds actual size.)

liferation of the glands is apparent, but there is no evidence of malignancy (Figs. 2, 3).

In this case there had developed, without discoverable cause, non-malignant tumorous nodulations in the endocervix, characterized by glandular hyperplasia and multicystic degeneration, with the production of partial obstruction of the uterus.

Case 3. E. J., aged 44 years, married 21 years, entered Passavant Memorial Hospital July 23, 1934. There had been an appendectomy, a removal of both fallopian tubes and one ovary and a resection of the other ovary, also a diagnostic curettage, in 1919, in St. Paul. The following year, having moved to Chicago, the patient was given local treatments for relief of persistent leucorrhœa. These treatments, administered at various times during 1920, included endocervical applications of tincture of iodine and of silver nitrate of varied strength. It is recorded also that the patient suffered from pelvic discomfort, and a semifluctuant mass was palpated in the region of the right broad ligament.

Ten years later, in 1930, when I first saw this patient, there was persistent pelvic pain, evidently from adhesions, and a fist sized soft mass was still present in the deep right pelvis. At the next visit, in 1934, immediately before entrance into the hospital, marked pelvic distress was still present. The patient gave history of regular menstruation every 28 days of 4 days' duration until the preceding year, since which time the periods had become slightly irregular but continued to be normal in amount and painless. Two months prior to entrance, immediately following a menstrual period, there had been a sudden gush of yellow fetid discharge. This was profuse for 3 or 4 days, followed by moderate, irritating, dark watery discharge which persisted until entrance into the hospital.

Operation. The appendix, tubes, and ovaries were found to be absent except for a tarry mass in the right broad ligament in which were some remnants of ovarian tissue. Massive pelvic adhesions almost completely concealed a heavy dragging uterus, to the right of which was the broad ligament hæmorrhagic accumulation just referred to. A complete abdominal hysterectomy was performed and the associated lesions were attended to.

The removed uterus was of generous size, 3 inches in length, the peritoneal surface studded with fibrous tags, the tubes absent. At the time of opening the uterus the external os was found to be stomatous, the cervical canal for ¾ centimeter above was narrow, granular and rigid, passing over suddenly into a sacculated space containing prune juice material. The endocervix in the sacculated region was rough and furrowed (Fig. 4).

Microscopic study of the pocketed rough endocervical wall above the strictured region reveals extensive glandular hyperplasia, the columnar epithelium of the glands having undergone extensive metaplasia into squamous epithelium. In some regions the squamous epithelium is seen replacing the columnar cells, in other regions the process is complete and the squamous layer has attained massive thickness. There is a marked tendency to proliferation but no evidence of invasion (Figs. 5, 6).

In this case the stricture of the lower cervix is evidently ascribable in part to infection, in part to instrumentation and local medication. Above the strictured, incompletely obstructed lower cervix, the epithelium of the pocketed and dilated endocervix, subjected to the irritation of accumulated menstrual fluid and débris, has undergone marked leucoplacic metaplasia into squamous epithelium.

Case 4. M. C., aged 27 years, unmarried, entered Passavant Memorial Hospital October 22, 193[illegible]. She had been seen 5 years previously, when a tentative diagnosis was made of chronic appendicitis and early endometriosis. The uterus at that time was sharply anteflexed. The patient then wandered elsewhere and the appendix was removed through a McBurney incision; the pelvis was not explored.

Upon her return to me the patient stated that for the past year there had been continuous pelvic discomfort. Menstruation was fairly regular, of a gushing character, excruciatingly painful, occurred at intervals of 23 to 25 days and was of 7 days' duration, muddy during the final days. There had been persistent dark brown menstrual flow for an entire month prior to entrance. For many months the

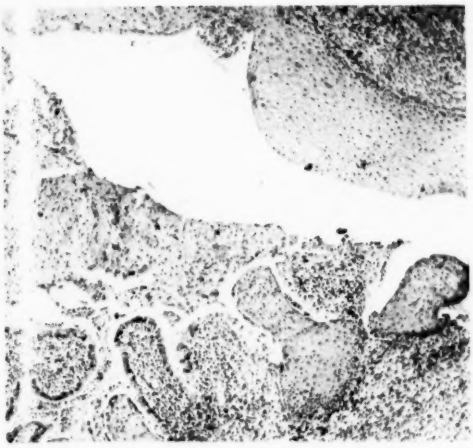


Fig. 5. Case 3. Photomicrograph of endocervical wall above stricture (see Fig. 4), revealing leucoplacic metaplasia of columnar epithelium into squamous epithelium. (The cavity in the illustration is a fold, not the cervical canal.) × 50.

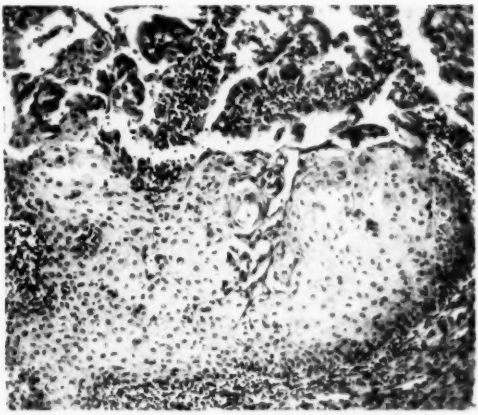


Fig. 6. Case 3. High power photomicrograph revealing squamous cell metaplasia of endocervix of uterus shown in Figure 4. × 90.

had also been a watery mucoid leucorrhœa, associated with cramps, beginning a few days after menstruation and continuing for 3 or 4 days.

Examination revealed the uterus somewhat fixed and huddled in retrocession, the fundus curled anteriorly, as in congenital anteflexion. Nodulations in the cul-de-sac and in the regions of the adnexa were pathognomonic of endometriosis.

At operation a textbook picture of extensive pelvic endometriosis was encountered. It was noted that the fundus of the retrocessed fixed uterus pointed anteriorly and certainly had never been retroflexed. Tarry cysts were present in both enlarged ovaries; "blueberry" and "raspberry" nodules and endometriotic adhesions completed the picture (Fig. 7).

The opened uterus had a rigid stomatous canal in the lowermost centimeter with a bell shaped endocervical pocket immediately above. The endometrium of the body of the uterus was succulent and numerous small polyps were present.

In this patient with extensive pelvic endometriosis and an upright uterus, a cervical stricture had apparently produced dilation above and retrograde spilling of menstrual débris and was the probable cause of the endometriosis.

Microscopic study of the pocketed endocervix at a level of a centimeter and more above the external os reveals a surface covered with festoons of hyperplastic glands. In many areas the columnar epithelium of the glands is being replaced or has been replaced completely by squamous epithelium. The squamous layer often attains a considerable thickness, but there is no evidence of invasion or other changes characteristic of malignancy (Figs. 8, 9, 10).

The pocketed endocervix of this patient was undoubtedly bathed in retained secretion, just as in Case 3. And again we find extensive squamous cell metaplasia. From these cases it would appear that the pocketed mucosa above cervical strictures is peculiarly predisposed to metaplasia, and in this we find two features of special interest: (1) a possible source of the cells which give rise to squamous cell endocervical cancer, and (2) an irritating retained secretion which may be a stimulant to cancerous growth. It should be emphasized that we have noted repeatedly the peculiarly irritating qualities of retained fluid which escapes into the vagina and bathes the vulva.

Case 5. W. K., aged 38 years, married 4 years, nulliparous, entered Passavant Memorial Hospital July 30, 1934. Increasing pelvic discomfort and severe dysmenorrhœa were the chief complaints. Menstruation was fairly regular but prolonged and characterized by premenstrual spotting for a week, followed by the passage of clots with establishment of the flow.

At operation the ovaries were found to be converted into tarry cysts, the left containing approximately a liter of dark bloody fluid, the right one-fifth as much. The upright body of the uterus was of double normal size and contained multiple small fibroids.

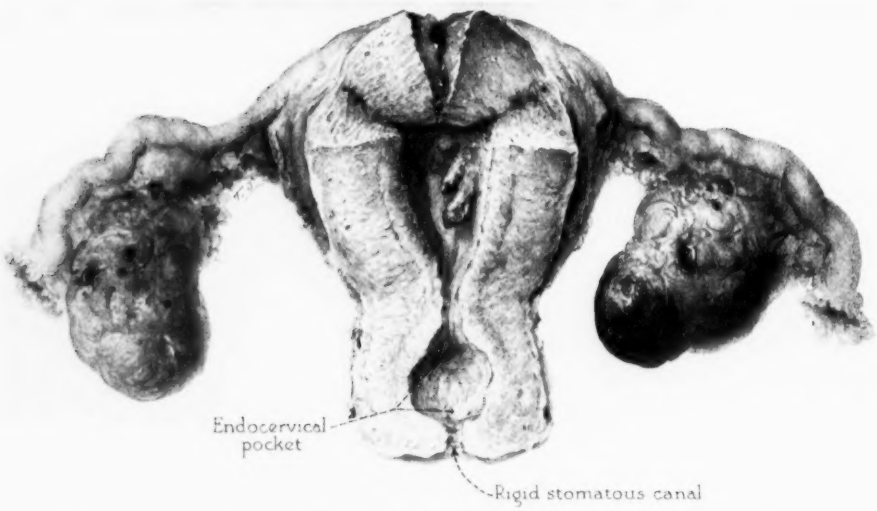


Fig. 7. Case 4. Pelvic endometriosis in a patient with an upright uterus, a cervical stricture, dilation above the stricture, squamous cell metaplasia of the endocervical columnar epithelium, and uterine polyps. Retained secretion above the stricture was probably a factor in producing the metaplasia; retrograde spilling of menstrual débris through the fallopian tubes may have caused the endometriosis. (Two-thirds actual size.)

The uterus was opened, after removal, and a granular endometrium was encountered. It was puckered but not definitely obstructed at the external os, and at the internal os was a rounded tumefaction similar to those found in Case 2, more than a centimeter in length, with associated puckering of the indurated mucosa and distinct narrowing of the lumen.

It is to be noted in this case, as in Case 4, that pelvic endometriosis developed despite the upright position of the uterus. And in conformity with my previous experience in patients with endometriosis and a well placed uterus, interference with uterine drainage was again encountered, the obstruction in this case being a cystic tumorous thickening at the internal os.

Microscopic study of the tumorous area reveals fibrotic tissue honeycombed with innumerable glands and cystic glands (Fig. 11).

It would appear that tumorous obstructions of the cervix (see also Case 2) may be composed of hyperplastic glands and stroma. This tissue is not bathed in retained secretions and seemingly there is no tendency to metaplasia of its columnar epithelium.

Case 6. R. S., aged 42 years, entered Passavant Memorial Hospital December 5, 1930. At the age of 29 years, the uterus had been dilated for relief of sterility, without result. There was no history of other instrumentation or of pelvic infection. Menstruation was normal and regular until 9 months before entrance, when it became excruciatingly painful and very profuse and of a brownish character much of the time.

Operation. Although there was a dark bloody discharge from the uterus, the cervical canal would not admit even the finest Hegar dilator. At abdominal hysterectomy the tubes and ovaries were found firmly plastered to the posterior surface of a uterus of double normal size which contained several small fibroids. The tubes were open at their fimbriated extremities.

Examination of the uterus after removal revealed cystic degeneration, stricturing and some pocketing throughout the length of the cervical canal, and stagnant dark liquid blood was present in the uterine cavity, evidently accumulated because of defective drainage.

In this patient without history or discoverable evidence of infection and no instrumentation other than dilation for sterility in her early married life, the etiology of the cystic degeneration and multiple firm strictures of the cervix is uncertain. Obstruction, with backpressure of the retained secretion and retrograde menstruation, rivals the more common infectious processes as the possible cause of the adhesions which held the patent tubes

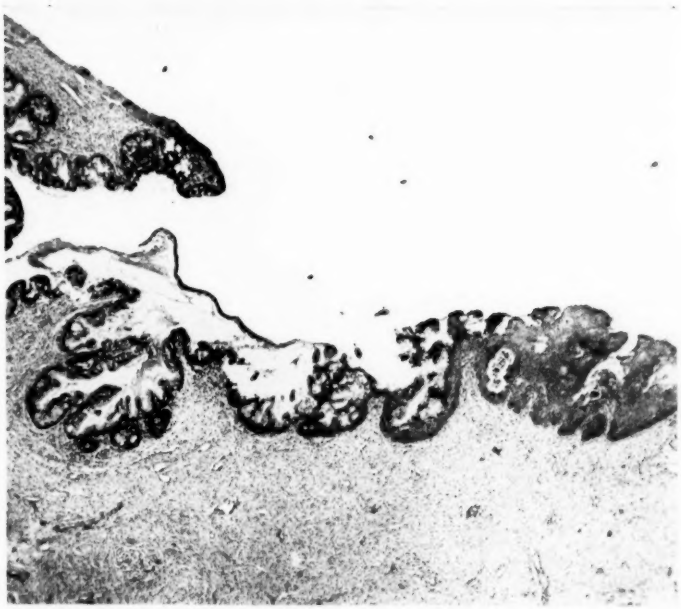


Fig. 8. Case 4. Low power photomicrograph of pocketed endocervix showing hyperplastic endocervical glands and metaplasia of columnar epithelium into squamous epithelium. × 31.

and the ovaries to the posterior surface of the uterus.

Case 7. M. M., aged 38 years, married 18 years, entered Passavant Memorial Hospital September 30, 1934. At the age of 22 years the patient had given birth to twins, delivered by forceps. A bilateral thrombophlebitis developed in the puerperium and was persistently recurrent thereafter. When 27 years of age she had received a radium treatment, evidently an intracervical 2 hour 50 milligram application, for a "scar in the cervix." At the age of 32 years another child was born, a hard labor with bag insertion, followed by a recurrence of the thrombophlebitis.

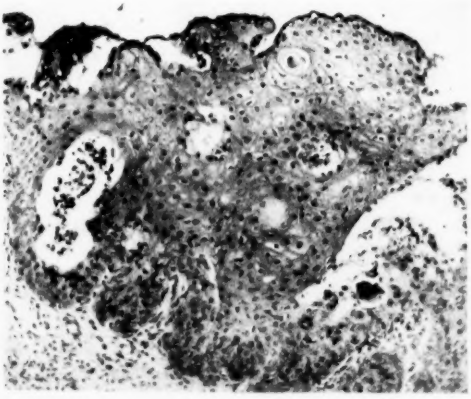


Fig. 9. Case 4. Higher power photomicrograph of area of metaplastic epithelium which is also shown in Figure 8. × 95.

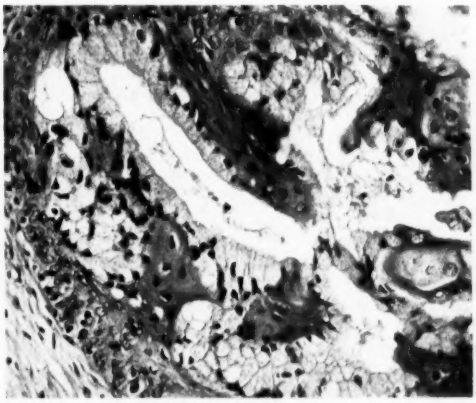


Fig. 10. Case 4. Photomicrograph of endocervical epithelium under higher power (see Fig. 8) showing deeply stained squamous cells replacing the columnar cells. × 190.

Fig. 11. Case 5. Low power photomicrograph of endocervical tumefaction, consisting of a stroma honeycombed with a multitude of glands and cystic gland spaces (see also Fig. 2). × 8.

At this time the patient came to me and was more or less under my care thereafter because of pelvic discomfort and lacerations. She was also attended by an internist because of intractable colitis and recurrent fever. Menstruation became irregular at the age of 36 years, 2 years before entrance into the hospital. For 6 months the flow had been very profuse, greatly prolonged, and accompanied by severe dysmenorrhœa. A persistent, dark brown, tarry discharge was present in the intervals.

Surgery was finally resorted to not only because of bleeding and progressive increase in the size of the uterus but also with the hope that operation might help the colitis. At exploration a varicocele nearly the size of one's wrist was found in the left broad ligament; a mass of veins one-third as large was present on the right, one of them distended with an old thrombus.

The removed uterus was more than twice normal size and measured 6¼ inches in length. The external cervix was badly lacerated and frayed; bisection revealed that the higher endocervix had undergone extensive cystic degeneration, the cervical canal being reduced to an exceedingly narrow distorted channel, barely admitting a probe. The constricted canal of the cervix passed over abruptly into a spacious uterine cavity. The body of the uterus presented a diffuse adenomyosis with massive thickening of the wall throughout (Fig. 12).

In the dilated pocketed lowermost portion of the uterine cavity we again found extensive metaplasia (Fig. 13). A rugged columnar epithelium was well preserved in some areas; in others it was more or less completely replaced by squamous cell metaplasia which often attained a depth of numerous layers. In the deeper uterine wall in this region, as well as elsewhere, there was extensive adenomyosis.

From clinical observation of the progress of this case as well as from study of the pathology encountered, I am led to believe that stricture of the cervix with backpressure of retained secretions and menstrual blood was the cause of the adenomyosis of the uterus as well as the squamous cell metaplasia of the endometrium.

Case 8. M. M., aged 49 years, unmarried, entered Passavant Memorial Hospital January 13, 1933. Regular menstruation during early years became more frequent and prolonged, changing to an irregular excessive flow at the age of 46. At the time of entrance there had been an amenorrhœa for 7 months followed by profuse, very dark bloody discharge which had persisted for 3 weeks.

Despite negative evidence on palpation of this virgin patient, exploration of the uterus appeared necessary.

Operation. On dilation and curettage many pockets were encountered within the uterus and much succulent material was obtained. Because of multiple strictures and a suspicion of malignancy a vaginal hysterectomy was then resorted to. The tubes were found to be patent, but both tubes and ovaries were adherent and covered with innumerable superficial cystic nodules; the left ovary was largely replaced by a retention cyst. The uterus, opened after removal, was rigidly puckered and constricted both at the external os and at the internal os, with an intervening endocervical pocket the size of a walnut, the posterior cervical wall being thinned out and herniated. A polyp protruded into the cavity of the cervix. At the internal os the mucosa was rigid and corrugated, and the endometrial cavity above contained slimy dark fluid and innumerable polyps projecting into the lumen from the œdematous moist mucosa (Fig. 14).

The microscopic structure of a broad based polyp at the right uterine horn revealed a papillary malignant growth.

Pathological diagnosis: Multiple strictures in a virgin uterus. Retention and back pressure of secretions with (1) endometrial polyposis developed in the moist uterine cavity, one growth in the right cornua presenting a picture of early papillary adenoma malignum, and (2) innumerable minute peritoneal cysts and slight adhesions of the tubes and ovaries.

In this case of obscure etiology, occurring in a virgin patient, obstruction and retention of secretions evidently predisposed to endometrial polyposis. One of the polyps presents a tendency to abnormal proliferation. Re-

gra le spilling from the patent tubes may account for the adnexal pathology.

CASE 9. A. G., aged 40 years, married 16 years, entered Passavant Memorial Hospital January 3, 193 . There was a history of 2 births, one a forceps delivery, with uncomplicated recovery after both. Two therapeutic abortions had been performed because of toxæmia of pregnancy. Subsequently, in 192 , there was a pelvic operation with removal of adherent tubes, suspension of the uterus, and a vaginal trachelorrhaphy. Dysmenorrhœa, present since puberty, became more marked during the year following this operation. Premenstrual cramps of several days' duration became excruciatingly severe 6 months before she came to me, in 1933, and the menstrual flow, established with difficulty, became prolonged and profuse; the history was typical of uterine obstruction.

At operation extensive adhesions were encountered throughout the abdomen, being most marked in the pelvis. The right ovary was cystic and adherent, the left normal; the tubes were absent. The large uterus was buried in adhesions.

The uterus was opened after removal, and it was found that amputation of the cervix at the previous operation had resulted in a snug fibrotic closure at the reconstructed external os, with some pocketing immediately above. In the vicinity of the internal os there was a ragged polypoid thickening of the endometrium with superficial necrosis. The involved area included nearly the entire circumference just above the internal os and was 1 centimeter in length; it apparently obstructed the lumen. A grossly recognizable wedge of invasion into the musculature suggested the possibility of malignancy. There was no dilation of the uterine cavity above (Fig. 15).

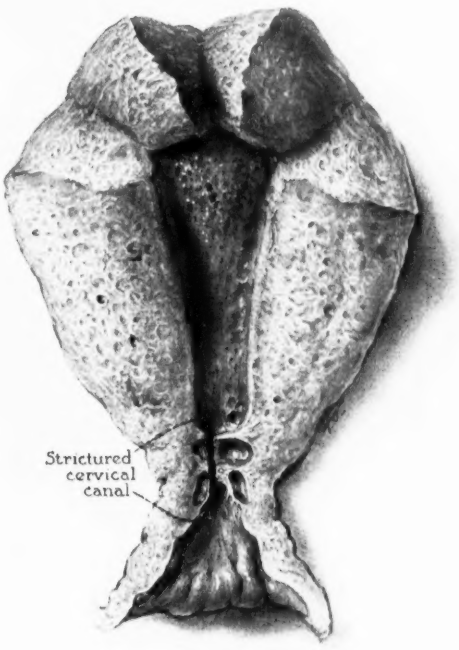


Fig. 12. Case 7. Massive uterus, 6¼ inches in length, with a lacerated frayed external cervix and a cystic badly strictured inner cervix terminating abruptly in a spacious uterine cavity, the body of the uterus presenting diffuse adenomyosis. (Two-thirds actual size.)

Fig. 13. Case 7. Photomicrograph of endometrium from lowermost portion of uterine cavity of uterus in Figure 12. Squamous cell metaplasia of columnar epithelium. × 100.

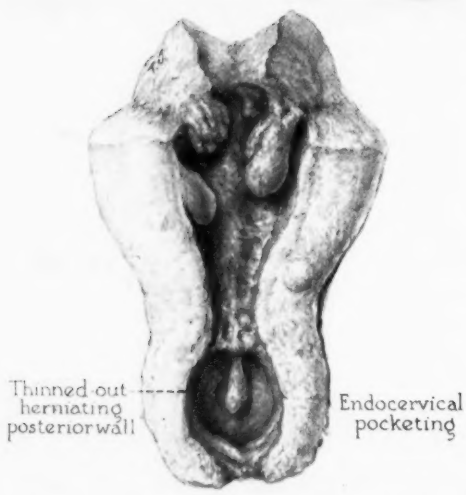


Fig. 14. Case 8. Virgin uterus with dense strictures at the external os and the internal os, a dilated pocketed endocervix, accumulated fluid in the uterine cavity, and endometrial polyps. Microscopic examination of the broad based polyp at the right uterine horn reveals a papillary adenoma malignum. (Two-thirds actual size.)

Microscopic study revealed marked cystic degeneration of the tissues in the region of fibrotic narrowing in the lowermost cervix. The tumorous growth above the internal os proved to be an early adenocarcinoma.

In this case a fibrotic narrowing at the external os apparently developed after a rather low operative repair of the cervix. Whether a polypoid growth occurred at the internal os because of retained secretions and a cancer developed in this region incident to and because of a benign tumorous obstruction, or whether the malignancy occurred primarily and independently is a question. I assume that obstruction at the external os predisposed to the polypoid growth above; whether the latter was primarily benign or malignant is problematical.

Case 10. E. F., aged 27 years, married 7 years, never pregnant, reported for examination November 5, 1926. The uterus had been curetted 2 years previously and a year thereafter a Rubin test had been made; it was not certain whether the tubes were open.

When the patient came to me she complained of severe dysmenorrhœa of 2 months' duration. The uterus was well placed and no pathology was demonstrable on palpation, but a stricture was found in the cervical canal. It was dilated with much difficulty. A Rubin test demonstrated the patency of the fallopian tubes. The fibrous obstruction of the cervical canal was overcome by use of an electric cautery, but the obstruction recurred repeatedly during the next several years. Dysmenorrhœa of increasing severity became complicated by pelvic discomfort at other times and eventuated in a typical clinical picture of pelvic endometriosis.

At operation, September 15, 1934, the right ovary was found converted into a tarry cystic mass of four times normal size and extensive endometriomatous lesions were scattered elsewhere throughout the pelvis. The cervix was firmly strictured, the uterus in excellent upright position.

This patient evidently developed extensive diffuse pelvic endometriosis during the period that she was under my observation. The upright position maintained by the uterus throughout this time is a feature of interest. A persistently recurrent stricture of the cervix evidently created back pressure and retrograde menstrual spill into the pelvis and presumably was the cause of the endometriosis.

Case 11. H. P., married, now 32 years of age, has been under my care since a girl of 18, when she reported with a virulent gonococcal infection of the lower genital tract of 1 month's duration. The disease localized chiefly in the cervix and treatment was directed toward eradication of this focus upon subsidence of the active infection. The measures employed consisted of the usual procedures then in vogue—topical use of iodine and silver nitrate introduced on applicators, applied as gently as possible to the lowermost cervix, and later, with failure of improvement, to the endocervix at a higher level. The cervical canal became very tight and dilations were resorted to at various times. The stubborn persistence of a profuse creamy discharge led eventually to the intracervical use of radium. Occasional dilations of the cervix were required for the relief of distress from obstruction during the next 2 years but the symptoms gradually disappeared.

In 1925 a Rubin test revealed patency of the fallopian tubes. The uterus remained in an upright position, the adnexa apparently normal.

Two years later symptoms of cervical obstruction again recurred and the nasal tip cautery was applied to the endocervix under gas anæsthesia.

There followed a period of 4 or 5 years of relative comfort. Menstruation was scanty but apparently without obstruction. Then the patient began to have scanty, irregular menstruation with cramps and a discharge of dark menstrual blood. Pelvic distress increased and small fibroids were palpable in the uterus.

Operation, April 10, 1934. Endometriomatous adhesions were encountered in the cul-de-sac. Firm bands held the sigmoid and rectum to the body of

the uterus, which was well placed and upright. The ovaries were small and healthy except for superficial endometriosis of the left.

The removed uterus contained multiple small fibroids and was atrophic. The cervix, not more than half the usual size, was strictured at the external os, and in a pocket immediately above was ½ cubic centimeter of fetid tarry blood. At the internal os was another stricture, but no fluid was present in the endometrial cavity. The endometrium was atrophic. There were many small fibroids in the wall of the uterus, the largest the size of a plum.

This patient, attended by me since she acquired a gonorrhœal infection in her youth, apparently developed a cervical stricture as the combined result of a severe infection and endocervical treatments directed toward eradication of the infection and relief of a persistently recurrent endocervical obstruction. Blocked drainage from the uterus *may* have been a factor in the etiology of the uterine fibroids, and retrograde spilling of menstrual fluid was a probable factor in the development of pelvic endometriosis, which occurred despite the upright position of the uterus.

CASE 12. B. C. Six years ago, at the age of 27, this patient, married two and one-half years, reported to a physician for examination because of a missed menstrual period and suspected pregnancy. An erosion was found and an electric cautery knife was applied to the cervix.

When first seen by me 9 months later there had been persistent amenorrhœa since the time of cauterization. The cervix was closed tightly, the body of the uterus was soft and several times normal size.

At operation the cervix was found to be completely strictured, the entire uterus converted into a soft boggy mass, the walls of the cervix thinned out to a thickness of a few millimeters. Two hundred cubic centimeters of dark liquid blood escaped upon opening the cervical canal.

The fallopian tubes were tested for patency 3 months later and were found to be obstructed, but the obstruction was readily overcome. Sterility has persisted, but pelvic discomfort, which caused considerable distress for a year or 2, has gradually disappeared.

Strictural obstruction of the cervix required repeated dilations during the first 3 years following drainage of the hæmatometra, but the canal has now remained patent for several months without dilation, although occasional use of a Hegar dilator is still necessary. Menstruation is scanty and there is evidence of an impending premature menopause at the age of 33 years.

Other cases of hæmatometra occurring in early married life have been encountered. It

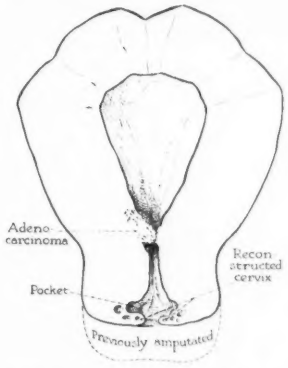


Fig. 15. Case 9. Diagrammatic illustration of uterus with a reconstructed cervix pocketed above, incidental to repair. There was gradual development of a tight external canal, with back pressure retention. Early adenocarcinoma just above internal os. (One-half actual size.)

is not a rare complication following avulsion of the cervix during delivery. Re-establishment of a satisfactory canal is exceedingly difficult unless corrected within a few months. Sterility is the rule. Retention of menstrual blood with complicating pelvic pathology has made surgical intervention necessary in the majority of cases which I have encountered.

SUMMARY AND CONCLUSIONS

1. The frequency of cervical strictures merits repeated emphasis; they are of common occurrence in the practice of everyone concerned with pelvic diseases of women.

2. The various therapeutic procedures employed in treatment of lesions of the cervix are more important than any other factor in the etiology of cervical obstructions. Endocervicitis is also important in the etiology, and in the majority of cases of serious stricture there is a history of an inflammatory process as well as of instrumentation. It must not be assumed, however, that absence of a history of infection or of instrumentation invariably gives assurance of a patent cervical canal and adequate uterine drainage. Strictures of the worst kind are encountered in virgin patients and in others in whom there is no discoverable predisposing factor.

3. Fibrotic constriction of the cervical lumen is much more serious than simple obstruction by adhesions. If drainage is not free,

pocketing of the cervix may occur immediately above the fibrous obstruction.

4. A metaplasia of the columnar epithelium into squamous epithelium has been found in the roughened pocketed *endocervix* above cervical strictures. A similar metaplasia has occurred in the pocketed *endometrium* above a cervical stricture. Irritation from retained secretions may be a factor in stimulating the metaplasia. (The question arises as to whether the seepage of irritating retained secretions from the pocketed cervix may be a factor in the production of leucoplacia of the external cervix.) We find here, in the presence of leucoplacic squamous cell metaplastic tissue bathed in retained menstrual fluid and débris, a possible explanation of the frequent incidence of squamous cell cancer of the endocervix and adjacent endometrium.

5. Tumorous nodulations of the endocervix and lower uterine segment are a less common cause of uterine obstruction.

6. The effects of damming back of uterine secretions and menstrual blood may be far reaching—perhaps important in the etiology of endometrial polyps, adenomyosis of the uterus, pelvic endometriosis, and back pressure inflammatory processes in the pelvis. The relationship of obstructive retention to the development of intra-uterine cancer is a problem which merits further study.

7. If my individual experience is a criterion, it will eventually be regarded as a surgical sin to use radium, or make repeated electrocervical topical applications, or apply the cautery within the cervical canal, or amputate the cervix of the uterus, or even administer expectant care to a patient with notable endocervical disease, without prolonged and attentive observation thereafter directed to the maintenance of patency of the cervical canal. Our thoughts have been too little concerned with the lumen of the uterus and the importance of free uterine drainage.

THE PREPERITONEAL LAYER—ITS GYNECOLOGICAL APPLICATION

JOSHUA WILLIAM DAVIES, A.B., M.D., F.A.C.S., NEW YORK, NEW YORK
From the Department of Anatomy, Columbia University Medical School and the Woman's Hospital, New York

SUPERFICIAL to the peritoneum, there is a definite layer of fibro-areolar tissue containing varying quantities of fat, smooth muscle, and elastic tissue. It is this structure which disorients the student about to make an incision into the abdominal cavity, especially in the midline.

Various names have been given to the layer. The term fascia endo-pelvina is confusing unless one adopts the classification in which two systems of fascia are recognized: the one, a dense supportive tissue applied to voluntary muscle and the other, an areolar fascia applied to abdominal and pelvic organs. Gallaudet called this layer the subperitoneal fibro-areolar layer, due to the fact that it is found beneath the peritoneum when the operator is working within the abdominal cavity. Others call it the extraperitoneal layer, which in itself is incomprehensive because the muscular as well as the subcutaneous and skin layers are likewise extraperitoneal. The Germans refer to the layer as *Bindegewebe*, i.e., connective tissue, because it is applied to and binds the various structures of the abdomen and the pelvis. The term "preperitoneal" accurately localizes the structure in the abdominal wall.

In the abdominal region the layer is closely related to the peritoneum as it ensheathes the adrenals, kidneys, ureters, vas, colon, vessels, and nerves; but as it extends into the false and true pelvis to ensheath the rectum, vagina, bladder, seminal vesicles, and the prostate, it is apparently not so intimately connected with the peritoneum. It does, however, continue to lie between the peritoneum and the transversalis fascia, which in the pelvis is called the supra-anal fascia. During youth the general tonicity and the deposit of fat aids in maintaining the supporting function of this structure, but illness with the absorption of fat, or old age with the diminishing of general tonicity predisposes to abdominal and pelvic relaxations. During the childbearing period, nature attempts to restore the relaxed or lacerated structure by increasing the proportion of involuntary muscular and fibrous tissue, thereby postponing the fatiguing symptoms of prolapsus until the support of pelvic congestion is removed by the menopause.

Considerable discussion exists as to the nature and the origin of the structure surrounding the vagina, uterus, bladder, and the rectum. Each of these structures must perforate the musculo-fascial layers of the pelvic outlet to function normally and consequently their covering must fuse with the transversalis or supra-anal fascia, covering the superior surface of the levator ani muscle. To assume that distensible organs are covered with a definite restraining structure such as that covering voluntary muscle is highly improbable; but by beginning in the abdominal wall and tracing the preperitoneal tissue around the adrenals and kidneys into the false and true pelvis in a single sheet, one can more readily understand the consistency and function of the structure without the use of a microscope. Histologically proving a structure does not interpret its relationship.

Pelvic surgeons are familiar with the terms, pubocervical, paravesical, paravaginal, pararectal, presacral, uterosacral, lateral ligaments of the vagina, and pillars of the rectum, but many fail to see that they are merely thickenings of the preperitoneal layer, especially so around the vessels and nerves as they course from the lateral pelvic wall to the pelvic organs. It was because of a desire to comprehend this structure that this study was carried on.

In Figure 1 the peritoneum has been removed from a male specimen thereby exposing a continuous layer of preperitoneal tissue which extends from the abdominal into the pelvic region. It will be noted that the ureter, abdominal vessels, and nerves are ensheathed and supported by this structure. As they are traced to the rectum and the bladder, it is noted that the same layer envelops them

Fig. 1.

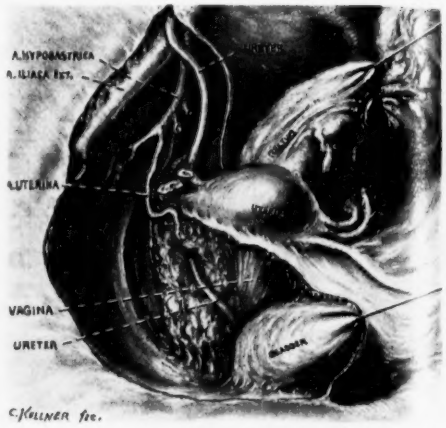


Fig. 2.

thereby protecting and aiding in maintaining their position. On the left, the layer of preperitoneal tissue has been incised and retracted to expose the transversalis fascia covering the psoas muscle. Anteriorly, retraction of the layer exposes the bladder, vas, and seminal vesicles. In the region of the seminal vesicles the involuntary muscular fibers of the layer are increased and probably aid in compressing the seminal vesicles during ejaculation, since they are devoid of a muscular coat.

After removal of the coccyx and the lower segments of the sacrum preparatory to the removal of the rectum for carcinoma, it is necessary to incise the preperitoneal layer before the rectum can be exposed. This layer has been called the presacral layer. In the same manner it is necessary to incise the preperitoneal layer to expose the seminal vesicles, prostate, or bladder. With the Latzko cæsarean section, the layer is divided by blunt dissection and the bladder is retracted to expose the anterior surface of the uterus.

From the anterior surface of the rectum the layer can be traced anteriorly to the bladder. An incision in this region will expose the continuation of the transversalis fascia on the superior surface of the levator.

To facilitate a comprehension of the structure in a female, let us interpose the vagina and the uterus between the rectum and the bladder and at the same time perforate the levator and its fascial coverings, thereby establishing a communication with the vulva. The vagina and the uterus will then be ensheathed by the same layer as the rectum and the bladder, from which it receives support, and especially from the thickened strands of involuntary muscular and fibrous tissue contained therein.

In Figure 2 the peritoneum on the right side of a female pelvis has been removed thereby exposing the preperitoneal layer about to be studied. By blunt dissection, the layer was mobilized from the lateral pelvic wall as a wide sheet, thereby exposing the continuation of the transversalis fascia into the false and true pelvis. It is noted that this procedure carried with it the contained rectum, vagina, uterus, and the bladder.

In Figure 3 the peritoneum was separated from the cul-de-sac, thereby isolating a distinct layer of preperitoneal tissue which lines the pelvis. Anteriorly, within the layer, is the bladder; posteriorly, the rectum; while medially are the vagina and the uterus. The blood supply to the rectum comes from the superior and middle hæmorrhoidal vessels which traverse this structure and, except for the missing peritoneal covering, is a structure not unlike a mesentery. The blood supply to

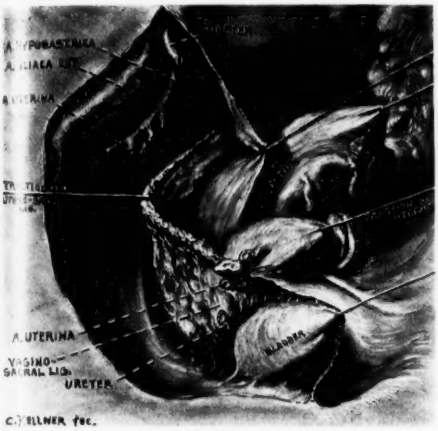


Fig. 3.

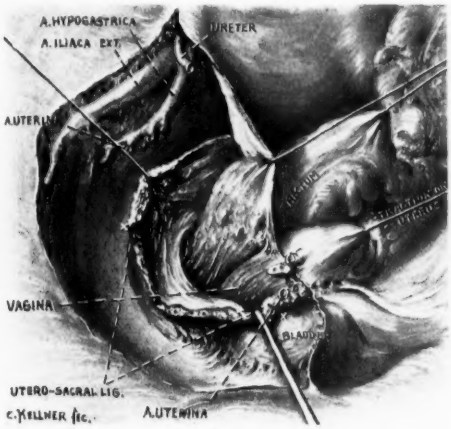


Fig. 4.

the bladder is from the superior, middle, and inferior vesical vessels which run in the preperitoneal layer from the internal hypogastric trunk to the lateral wall of the bladder. This organ can be then considered to be the center of a sling composed laterally of blood vessels and nerves contained within the preperitoneal layer.

The component parts of the uterus and the vagina, the muellerian ducts, develop in the layer midway between the rectum and the vagina where they are supplied by branches from the internal hypogastric vessels. As they approximate each other and fuse, the uterus and the vagina are formed. They are likewise covered by a layer of thin preperitoneal tissue and are supported laterally by its vascular supply, the lateral mesenteroids of the vagina. This term is used because the blood supply to the individual muellerian duct is not unlike a mesentery of the intestine, except that it is devoid of a peritoneal covering.

Stretching laterally and posteriorly from the uterus is the familiar uterosacral ligament which is composed principally of involuntary muscular strands and fibrous tissue. However, after the cul-de-sac is stripped of its peritoneum, it is noted that the uterosacral ligament, as such, is not a distinct free muscular strand like the round ligament, but it is the upper free margin of a distinct plane of tissue lining the true pelvis. That portion of the plane distal to the uterus is attached to the lateral wall of the vagina and may be called the vaginosacral ligament, or more descriptively, the lateral mesenteroid of the vagina.

In Figure 4 the uterosacral ligament and the vaginosacral ligament have been incised proximodistally to its distal boundary, the supra-anal fascia.

In Figure 5, that portion of the preperitoneal tissue distal to the cul-de-sac is shown. The posterior portion depicts the divided uterosacral and vaginosacral ligaments, thereby exposing the preperitoneal layer covering the supra-anal fascia between the rectum and the vagina. This layer contains branches from the middle hæmorrhoidal artery which are distributed to the posterior wall of the vagina.

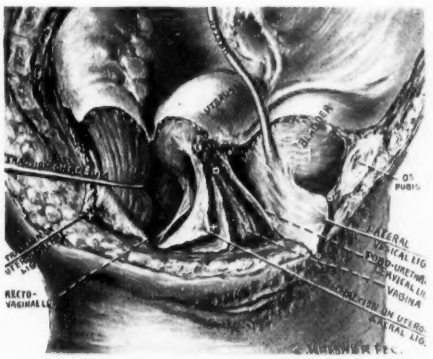


Fig. 5.

It has been called a pillar of the rectum, because of the definite hypertrophy noted during operations for the cure of a rectocele. Structurely it is the rectovaginal mesenteroid and when overstretched or lacerated by childbirth, the rectum is permitted to bulge the lower third of the posterior vaginal wall.

Anterior to the vagina, the vaginosacral ligament becomes continuous with the lateral vesical ligament which contains the vessels and nerves supplying the bladder. It has been separated from the lateral wall of the vagina and retracted anteriorly to show a structure similar to the rectovaginal mesenteroid, which extends from the pubis to the urethra and from the urethra to the cervicouterine junction. This structure is, however, rather intimately attached to the distal surface of the bladder and when relaxed or lacerated by childbirth throws an additional stress against the anterior wall of the vagina, which, if similarly relaxed, will result in the formation of a cystocele. It must be understood that the covering of preperitoneal tissue on the anterior and posterior walls of the vagina is of little value in the prevention of a cystocele or a rectocele, but the rectovaginal mesenteroid and the pubocervical ligament are distinctly efficient.

RETROVERSION

In the upright posture the uterus appears to be suspended by the uterosacral ligaments, while traction by the round ligaments and intra-abdominal pressure force the fundus anteriorly. Utilization of the round ligaments is more efficacious than the plication of the uterosacrals for the operative relief of this displacement. If, however, there is a tendency to prolapsus and a relaxation of the walls of the vagina, insufficient for repair, the plication or transplantation of the uterosacrals will be of value.

PROLAPSE

During the third and fourth decades of life a retroverted uterus is able to withstand the stress of intra-abdominal pressure, due to the support given to the uterus by its lateral attachments; however, with advancing years and a concomitant general relaxation, the lateral support permits a sagging of the uterus. An operation for retroversion together with a shortening of the uterosacrals and a tightening of the lateral ligaments will relieve the condition. However, as is usually the case, other plastic surgery is needed below; therefore, the plication of the lateral uterine ligaments, as suggested by Alexandroff, is sufficient, without opening the abdomen from above. The more extensive prolapse can be relieved by a vaginal hysterectomy. To obtain a deep vagina, and at the same time increase the tonicity of the pelvic outlet, the vault of the vagina should be closed from side to side, thereby tightening the sling of the vaginal mesenteroids, which support the vagina literally. This procedure, while improving the tonicity of the vault, will relax the uterosacral and the pubocervical ligaments unless definite steps are taken to unite them before the vault of the vagina is closed.

ENTEROCELE

A relaxation of the pararectal connective tissue, together with a deep cul-de-sac, permits a herniation between the vagina and the rectum which is evidenced as a bulging of the posterior wall of the vagina above the usual rectocele. The approximation of the uterosacrals from side to side will obliterate the cul-de-sac and act as an efficient barrier to a recurrence, if at the same time other necessary vaginal repairs are done.

RECTOCELE

Prolonged stretching of the vagina tends to prevent proper involution of the vagina and the surrounding structures, particularly the rectovaginal mesenteroid, which acts as an accessory barrier to pressure from the rectum against the lower third of the vagina, where the stress is more direct. The usual operative procedure in which the vagina is mobilized posteriorly and a triangular area of the relaxed tissue is removed, together with a repair of the pelvic floor is often times sufficient. If, however, the rectovaginal mesenteroid is first isolated and plicated in the midline or transplanted above the crest of the rectocele, as described by Dr. George Gray Ward, a more defensive barrier is established. An

tempt should be made to plicate or reattach this structure medial to the blood vessels therein, so as not to alter the vaginal blood supply.

CYSTOCELE

Prolonged stretching of the vagina, together with the eversion of the anterior wall by the descending head, tends to prevent proper involution of the anterior wall of the vagina and also the pubocervical ligament. Mobilization of the anterior wall of the vagina and the removal of the relaxed tissue restores the wall of the vagina and improves the tonicity of the lateral ligaments of the vagina, which in turn fairly satisfactorily relieves the cystocele; if, however, the relaxed or retracted pubocervical ligament, which is closely applied to the bladder, is plicated and restored in the midline, the tonicity of the bladder will be restored through the increased tension of the lateral vesical ligaments. To one's surprise, the removal of the redundant vaginal tissue will appear unnecessary, other than for the relief of the relaxed vaginal wall. Dr. William T. Kennedy has, likewise, repeatedly carried out this procedure. After a complete hysterectomy has been done the lateral ligaments of the vagina should be approximated in the midline and an attempt should be made to approximate the uterosacral and pubocervical ligaments over the vaginal vault, particularly if there is a concomitant cystocele. This procedure definitely restores the tonicity to the floor of the bladder.

No reference has been made to the repair of the pelvic floor which must necessarily be repaired, if the efficiency of the lateral ligaments of the vagina and the musculofascial layers of the pelvic outlet are to be utilized most effectively.

CONCLUSIONS

1. The pelvic cavity is lined by a layer of preperitoneal tissue similar to the lining of the abdominal cavity.

2. As the kidney is held by preperitoneal tissue, likewise are the pelvic structures supported.

3. Prolonged distention of the vagina causes relaxation of the vaginal walls, the pubocervical ligament, and the rectovaginal mesenteroid.

4. Following a complete hysterectomy, a deeper vagina can be obtained by closure of the vault from side to side, rather than anteroposteriorly.

5. A small cystocele can be treated from within the abdominal cavity by tightening the pubocervical ligament, or by reattaching it to the uterosacral ligament following a complete hysterectomy.

6. Rectocele follows injuries to the posterior wall of the vagina and to the rectovaginal mesenteroid.

7. Prolonged stretching of the vagina may relax the anterior wall as well as the pubocervical ligament which is rather intimately attached to the inferior surface of the bladder.

8. Reconstruction of the pubocervical ligament, in the midline, increases the support of the bladder through its lateral ligaments and makes the removal of the relaxed vaginal tissue appear to be an unnecessary procedure, other than to aid in the restoration of a normal contour.

9. A granulomatous infection of the posterior portion of the vulva or the posterior wall of the vagina may extend to the rectum by way of the lymphatics in the rectovaginal mesenteroid and cause a rectal stricture about 3 centimeters from the anus.

REFERENCE

1. DAVIES, JOSHUA WILLIAM. Abdominal and pelvic fascias with surgical applications. Surg., Gynec. & Obst., 1932, 54: 495-504.
2. Idem. The pelvic outlet. Surg., Gynec. & Obst., 1934, 58: 70-78.
3. GALLAUDET, B. B. The Planes of Fascia. New York: Columbia University Press, 1931.
4. WARD, GEORGE GRAY. Kelly's Gynecology. New York: Appleton, 1928.

DIVERTICULA OF THE DUODENUM

HAROLD C. EDWARDS, F.R.C.S. (ENG.), LONDON, ENGLAND
Surgeon, King's College Hospital; Hunterian Professor, Royal College of Surgeons

IN a woman of 80, dead of apoplexy, Chomel found a duodenal pouch containing 22 stones. This case, published in 1710, is the first reference to pouching of the duodenum to be found in the literature. The sac is described as an extension of the membranes *vers le haut de cet intestin*. Morgagni refers to Chomel's case in his "De Sedibus." The former thought the stones were gall stones, as the sac appeared to communicate with the common bile duct. In Morgagni's opinion these stones had passed through the bile duct and for some reason or other had been detained in the duodenum and had caused a diverticulum.

Morgagni mentions a case of diverticulum of the rectum, and gives a full account of diverticulum of the duodenum 2 inches beyond the pylorus, discovered postmortem in the body of a man, also dead of apoplexy. The English translation (1769) of Morgagni's description runs thus:

> . . . a kind of cellule, not very protuberating, but big enough to admit a finger, surrounded with no coat but the external one of the intestine, yet having not the least traces of any present, or past, ulceration in that part, as indeed there were not in the stomach, or the whole intestinal tube.

In 1796, Rahn describes what was presumably a duodenal diverticulum secondary to an ulcer, occurring in a woman of 22 who died from emesis. In 1815, Fleischman described 3 cases, all intimately related to the pancreatic and common bile ducts. Fleischman suggested as a cause, frequent distention with food, drink, or air. He also made reference to diverticula in the large bowel.

Habersohn in 1857 commented on the rarity of diverticula of the duodenum compared with those of the colon. He regarded them all as hernial protrusions of the mucosa through the muscular coat.

In the same year Harley described a pouch midway between the pylorus and ampulla of Vater in a man of 87. It contained a gall stone measuring 3 inches by 3¼ inches, and weighed 450 grains. The gall bladder was thick and shrunken and buried in adhesions, while the common bile duct was dilated.

The first comprehensive survey was made by Roth in 1872, who, after the study of 5 personal cases, concluded that diverticula of the duodenum are exclusively of the "false" variety; they do not get larger than a pigeon's egg and are usually empty. "False" is used, apparently, as opposed to "congenital."

In 1884, Turner contributed the first detailed description of the association between duodenal ulcer and duodenal pouches, and mentioned three varieties.

In 1893, Perry and Shaw made an extensive study of the postmortem records of Guy's Hospital, describing 13 cases of diverticula of the duodenum. These they subdivided into (a) non-inflammatory and (b) depending upon operation or outside traction. These authors formed the opinion that the development of the non-inflammatory pouches depends upon abnormally high pressure inside the duodenum, and their views in this connection will again be referred to.

The article of Perry and Shaw is the foundation stone of our present knowledge of the condition, and those interested in duodenal diverticula can do no better than to commence their study with this article.

Other publications follow in more rapid sequence; those of Fischer, and Rolleston in 1900, of Keith in 1910, Baldwin in 1911, Moynihan in 1912, and of Linsmayer in 1914. This last work is a second landmark. Linsmayer's observations are based upon postmortem material. He expressly set out with the intention of investigating the condition, and was surprised to find 45 examples of the simple (non-inflammatory) type and 4 of the secondary type in 1,367 postmortem examinations.

In 1915 appeared the first recorded case diagnosed by X-ray and verified by operation (Forsell and Key). During the war years, apart from a preliminary observation in 1916

on the X-ray diagnosis by Case, little of importance occurred. The year 1920, however, was epochal. Papers by Spriggs and Marxer in England, Case in America, and Clairmont and Schintz in Germany, described in detail the X-ray appearances of duodenal diverticula. Wilkie's well known publication followed in 1921. Many excellent articles, most of which are referred to in the following pages, appeared during the last decade, and a culminating point was reached in 1930 when Otto Hahn published an extensive "Review of the Literature of Duodenal Diverticula."

CLASSIFICATION OF DUODENAL DIVERTICULA

"Many instances of congenital intestinal diverticula of the duodenum have been recorded, particularly in the second portion. Now that screen examinations after a bismuth meal are so constantly made, duodenal diverticula have ceased to be looked upon as great rarities." This quotation from a recent exhaustive treatise illustrates the existing confusion with regard to the etiology of duodenal diverticula. The author has found that the vast majority of diverticula in this portion of the intestine are divided among the acquired hernial (or primary) type and those secondary to ulcer. The remainder are traction diverticula and anomalous forms. True congenital diverticulum—i.e., a diverticulum present from birth and identical in structure to the intestine from which it arises,—is a condition of exceptional rarity in the duodenum. This is a matter for surprise, for one would expect the duodenum to be a fertile source in view of the development of the pancreas from it.

The possibility of there being a congenital factor in the etiology of the hernial type of diverticulum is conceded (although not supported), but this does not entitle this type to be called congenital diverticula, for their morbid anatomy and their age incidence are diametrically opposed to the concept of a congenital status.

MATERIAL

The following account of acquired diverticula of the duodenum is based on the examination of the following material:

X-rays, 31 cases, of which 26 are probably of the hernial type (primary acquired diverticula) and 5 of the secondary type. Of these, 3 are secondary to ulcer and 2 due to traction.

Pathological specimens: 4 specimens of diverticula of the hernial type; 7 specimens of diverticula secondary to ulcer.

Operation cases. Three diverticula were exposed at operation, 1 of which was secondary to an ulcer, and 1 due to traction by an adherent gall bladder. The third was removed intact. (Fig. 7.)

THE RADIOLOGICAL STANDPOINT

Incidence. During the 7 year period 1925–1931, inclusive, X-rays have provided 31 examples of duodenal diverticulum. The total number of examinations made during this period was 4,631, so that in this series the percentage incidence is 0.67 per cent. The incidence of these cases in each year is as follows:

1925	1 in 521 X-rays—0.19 per cent
1926	1 in 580 X-rays—0.17 per cent
1927	1 in 630 X-rays—0.16 per cent
1928	6 in 653 X-rays—0.92 per cent
1929	9 in 612 X-rays—1.47 per cent
1930	7 in 781 X-rays—0.90 per cent
1931	6 in 854 X-rays—0.70 per cent

The jump in incidence from the 0.2 per cent (approximately) of the 3 years 1925, 1926, and 1927 to 1.0 per cent (approximately) of the succeeding years coincides with the remodelling of the routine X-ray technique in the early part of 1928, the chief added features being (1) screening of all cases, (2) examination in the oblique position. In the first 3 months of 1928, before the technique was thus altered, 1 case only was reported, but during the remaining 9 months 5 cases were reported. There can be no doubt that before 1928, cases of diverticula were being missed during examination, and the percentages for these years do not represent the true incidence of the condition. If the years 1929, 1930, and 1931 are taken, the figures are 22 cases in 2,247 X-ray examinations, or a shade under 1 per cent. This compares very closely with the figures of Andrews, 26 cases in 2,200 examinations (just over 1 per cent), and Spriggs and Marxer, who found, in their first series 10 cases in 1,000 X-rays (1.0 per cent). Case describes 85 cases in 6,847 X-ray examinations (1.24 per cent). Spriggs and Marxer in 1925, however, gave a

further series of 38 cases in 1,000 X-ray films, an incidence of 3.8 per cent, while Cryderman found 45 cases in 770 X-ray examinations (5.85 per cent).

Five of the author's 31 cases were definitely of the secondary type, 3 being associated with clinical and other radiological evidence of a duodenal ulcer, and 2 being in all probability traction diverticula. Another case, in which barium entered the hepatic ducts, and which will subsequently be described, is not included in these figures, as the exact pathology is undetermined. Five cases in which some doubt existed are also excluded. The figures in tabular form are thus:

Total cases	31
Primary	26
Secondary	
With ulcer	3
Traction	2

These figures probably bear no direct relation to the relative incidence of the primary type and the ulcer type. It is not always possible, even after screening and taking oblique roentgenograms, to detect an ulcer diverticulum. One sees a fixed deformity of the duodenal cap, but a diverticulum, though present, may be too shallow to show up clearly, or its position may be such that it does not show in relief. Thus in Case 32, operation revealed a well defined pouch on the anterior wall of the duodenum, that was wholly unsuspected by the radiologist (Fig. 1).

As opposed to the ulcer diverticulum, primary diverticula show up readily in a thorough X-ray examination. Confusion exists in some cases between a primary diverticulum and a dilatation of the common bile duct. It may be, in fact, impossible to distinguish between these two conditions—they may be associated in the same case—on radiological evidence alone.

Contrary again to the ulcer diverticulum, the primary type is detected more easily radiologically than at operation. The latter all arise from the second and third parts of the duodenum, and many lie behind, or are overlapped by, the pancreas. It is sometimes only with difficulty that a diverticulum can be found at operation even when its presence is known beforehand, and a definite search is made.

The same applies to postmortem examination, at which diverticula will easily be missed unless the examiner is alive to the possibility of their occurrence and opens the duodenum throughout its whole length, so that he may examine its concave margin carefully from the mucous membrane side

Radiological appearances. Duodenal diverticula are seen as smooth, barium filled pockets persisting at a 3 hour or 6 hour roentgenogram. Unless large and well defined a pocket in the first part of the duodenum associated with an ulcer will not be revealed. The duodenum may show marked deformity—often the shamrock-leaf deformity described by radiologists as typical of duodenal ulcer—but no definite diverticulum. Furthermore, the pockets secondary to ulcer are in most cases shallow and wide mouthed and there is no retention of barium in them. They empty with the duodenum.

On the other hand, in Case 32, a well marked ulcer diverticulum was seen without difficulty at operation (Fig. 1) but was unsuspected at X-ray examination. It is frequently impossible to establish the exact point of opening of a diverticulum into the duodenum, and a differential diagnosis between ulcer diverticulum and the primary type cannot readily be made. If, however, peristalsis is seen in the wall of the diverticulum, it is certain to be of the ulcer type. Diverticula of the second part of the duodenum, the commonest site for diverticula of the primary type, are readily seen by X-ray, and if the routine described above be followed, will rarely be missed.

Figure 2 is selected as a typical example. In this case the colon was also the seat of multiple diverticula. A partly filled, but well defined, pouch in the concavity of the duodenum is shown. It empties into the second part of the duodenum, and there was no retention of barium after 3 hours. The question of barium retention after the duodenum is empty must be carefully studied, as only in such cases can the diverticulum be suspected of causing symptoms.

Diverticula of the horizontal and ascending parts of the duodenum are rarer, and not so readily seen. This latter point may, in fact,

be a partial explanation of the rarity of this finding as compared with diverticula of the second part.

The oblique position for examination is of especial importance in revealing diverticula near the flexure, and increasing frequency of incidence of diverticula in this situation during the past 3 years is due in part to the adoption of this routine.

In diverticula of the horizontal portion, the mouth of the diverticulum is on the concave surface of the duodenum, and the sac in the great majority of cases passes upward in relation to the pancreas. With this dependent drainage, the barium may not enter the diverticulum, or if it does so, remain for so short a period that it escapes detection. In the rare cases in which the diverticulum has increased in size so much that it hangs downward, its presence is readily detected, for stasis will always be present.

Buckstein, writing in 1927, and before oblique views were adopted as a routine, recommended a duodenogram for showing up doubtful cases of diverticula near the flexure, in which position, he states, they may be confused with an ulcer crater on the lesser curvature of the stomach. A radio-opaque duodenal tube, the first 10 inches of which has many small perforations made in its wall, is swallowed until the duodenojejunal flexure is reached, as determined by examination under the screen. A thin suspension of barium is then slowly injected and an X-ray is taken immediately, so that an outline of the duodenum alone is obtained.

The oblique view has in our opinion, however, obviated the necessity for this elaborate technique, and the print published by Buckstein is not sufficiently convincing to warrant a trial of this method.

Traction diverticula are rare and difficult to distinguish radiologically from other types. Points to be noted in arriving at the diagnosis are the shape and fixation of the shadow. The diverticulum may not have a spherical rounded appearance but a somewhat conical outline. It will be fixed to neighboring viscera.

The most frequent sites for traction diverticula are in the gall-bladder region and at the flexure, where occasionally the intestine may become caught up by perigastric fibrosis around an ulcer of the lesser curvature of the stomach.

Identical X-ray appearances, however, may be caused by secondary inflammatory changes occurring in the primary form of diverticulum.

Pitfalls in X-ray diagnosis. Occasionally extra luminal flecks of barium in the first and second parts of the duodenum remaining after emptying of the gut will rouse suspicions of the presence of a diverticulum. A second examination should be made in such cases, and attention in screening carefully focused upon these abnormalities. Such shadows in the first part may be due to the presence of a small ulcer crater, and in the second part to barium getting into a dilated common bile duct, or to the anatomical variation of the opening of the bile and pancreatic ducts into the duodenum. In cases in which a smooth rounded appearance of the shadow cannot be obtained on palpation of the duodenum, a confident diagnosis of diverticulum cannot be made. Five such cases occurred in the series of X-ray plates under review (not included in the 31 cases of diverticula).

Anatomical variations in the duodenum may give rise to some difficulty in the diagnosis. Heacock describes a case of pseudo-diverticulum due to a fixed redundancy of the duodenum, without any defect in the wall of the intestine. This condition was confirmed at operation. Larimore also draws attention to this appearance. It is a degree of that duplication of the duodenum described first by Wilkie. In these cases, the longitudinal plica, which terminates at the ampulla, stands out as a well marked fold dividing the second part of the duodenum vertically into two compartments. The condition is undoubtedly a minor variation of complete duplication of the duodenum described by Calder, in which two definite tubes existed. Fairland described such a duplicature of the whole tract from the pylorus to the cæcum. Wilkie also describes an infolding of the duodenal wall in the transverse axis, giving rise to a ring-like constriction. He thinks it is a mild form of the duodenal stenosis met with occasionally in children.

These appearances are readily distinguishable from those of a diverticulum by careful screening, especially in the oblique view. The normal "feathery" appearance of the duodenum in duplicature is retained without interruption.

Other departures from the normal topography of the duodenum should not give any difficulty. Differences in shape, the enlarged bulb due to an annular pancreas, abnormal positions, and ileus are not readily to be confused. Shadows occasioned by other things, such as gall stones and pancreatic stones, will be excluded.

Unquestionably the most important procedure in X-ray diagnosis is the screening operation—palpation of the duodenum under observation. It transcends in importance the fixed roentgenograms, with which the clinician is furnished and outweighs any conclusions that may be deduced from these impressions.

PRIMARY ACQUIRED DIVERTICULA OF THE DUODENUM

Of the author's 5 specimens (4 postmortem and 1 operation), 4 are examples of single diverticula arising in relation to the entrance of the common bile duct (perivaterien). In the fifth, 2 diverticula are present, arising from the transverse part of the duodenum.

Specimen 1. The specimen illustrated in Figure 3 was obtained postmortem from a woman of 77 years, who died following an operation upon a carcinoma of the colon (Case 6). Diverticula were present also in the large intestine.

The duodenum has been opened by dividing the anterior wall in the midline. Bristles have been placed in the common opening of the common bile and pancreatic ducts in the ampulla of Vater and in the opening of the duct of Santorini above. The major ampulla is unduly prominent. Its right margin is continuous with a transverse plica which forms the anterior lip of a wide mouthed diverticulum, and which then continues as a well defined transverse plica on the posterior and right lateral wall of the intestine.

The direction of the diverticulum is to the left, and slightly upward and backward, following exactly the same course as the common bile duct through the duodenal wall. On the reverse side of the specimen, the diverticulum could be seen as an ill defined bulge of the duodenum into the pancreas. It is separated from the latter by a narrow interval filled with loose areolar tissue, and the pancreas could be freed by blunt dissection. The mucous membrane lining the pouch is smooth and free from rugæ, but otherwise normal. A microscopic section of the wall at the fundus of the diverticulum shows only a small number of muscle fibers lying in a thin wall of fibrous tissue. A large vessel is lying within the wall.

Specimen 2. The specimen illustrated in Figure 4 is from a dissecting room subject. The pancreas has been removed except for a small portion of the head in immediate contact with the concavity of the duodenum. This has been dissected free, and turned upward. Between the pancreas and the common bile duct there is a large diverticulum. This is flask shaped, but not completely regular in outline. This fact is due in part to irregular shrinkage following prolonged preservation in a formalin solution. The wall of the diverticulum is as thin as tissue paper, and transmits light equally well. Microscopic sections show the wall to consist of a very thin layer of fibrous tissue, with no trace of a muscle coat. The mucous membrane has been partially destroyed by autolysis.

When the duodenum was opened, it was found that the relation of the mouth of the diverticulum to the ampulla of Vater was similar to that in Specimen 1.

Specimen 3. The specimen illustrated in Figure 5 is from a dissecting room subject, a man of 79. The duodenum is an unusually large one, but no obstruction was present. The walls are of about normal thickness, there being no evidence either of thinning from passive distention, or thickening from hypertrophy. A small portion of pancreas remains attached to the duodenum, and is retracted forward by a hook. Between this portion of the pancreas, and above and in front of the common bile duct, is a large diverticulum. It has been packed out with cotton wool, but before this was done was lax and collapsed. The wall is extremely thin, and readily transmits light. The mucous lining is smooth, and contains no rugæ. The diverticulum in the recent state lay posterior to the duodenal margin of the head of the pancreas, from which it was separated by a thin layer of areolar tissue.

The relation of the mouth of the diverticulum to the ampulla of Vater is as in Specimens 1 and 2.

Specimen 4. This specimen, illustrated in Figure 6, was obtained in the dissecting room from the body of a man aged 71. To obtain the illustration, the specimen was suspended, and viewed from above and behind. A portion of the head of the pancreas remains attached. Two small diverticula are present, opening from the transverse portion of the third part of the duodenum, the second one (nearest the "camera") being at the junction of this part with the ascending part of the duodenum. The double row of blood vessels supplying this part of the duodenum, to be described in full subsequently, is clearly seen.

The outline of this diverticulum is indicated by an interrupted line, as it was removed flush with the

intestine before the drawing was made. Whole serial sections were cut, one of them passing through the maximum girth of the diverticulum.

The double row of blood vessels supplying the duodenum, to which a detailed reference will be made later, is clearly seen, one row lying posterior and the second anterior to the midline. The latter is represented by the thick interrupted line in the illustration.

In each case, the opening of the diverticulum is to the posterior side of the midline, in relation to one of the posterior row of blood vessels. This is very clearly shown in the smaller diverticulum, for the section through its base has cut through an artery, which ran into the wall of the diverticulum, and is well shown in the section.

Microscopic appearance. A section passing through the maximum diameter of the diverticulum, which is completely devoid of a muscular layer, shows that the muscle of the duodenum ceases abruptly at the mouth of the diverticulum, and there is no suggestion of it being carried along in its wall. The muscle in the angle between the intestine and the pouch appears to be a little thicker than elsewhere in the duodenum, as though it had undergone a certain amount of hypertrophy. A large artery can be seen between the submucous layer of the diverticulum and the muscularis of the duodenum, and its companion vein is seen as a long narrow chink. This artery corresponds with the one cut across, as shown in the illustration. The diverticulum consists of an extremely thin layer of fibrous tissue, covered with serosa, and lined by mucous membrane; the latter is thin and contains no rugæ. No pancreatic tissue or Bruenner's glands are present.

Specimen 5 (*Fig. 7*). Perivaterien diverticulum removed at operation from a woman of 54 years. It is a thin walled sac, devoid of a muscular coat and having a cubic capacity of 14.5 cubic centimeters. The radiological appearance is shown in Figure 8. The patient complained of abdominal pain, with occasional sickness, and a distressing amount of flatulence after meals. The history extended back for some 9 or 10 years, and the symptoms had become progressively worse despite periods of treatment elsewhere for gastric ulcer. Six years before admission appendicectomy had been done, with no relief. No other cause for the symptoms was demonstrated, and removal of the diverticulum resulted in a complete cure of the distressing flatulence. The patient still complains of occasional pain, but this has very greatly improved, and 11 months after operation her weight had increased from 7 stone 6 pounds to 8 stone 10 pounds.

A typical primary duodenal diverticulum is thus a thin walled sac opening into the bowel by a wide mouth. Except in exceptional instances, it rarely exceeds the size of a golf ball. It is rounded in shape, as opposed to the finger-like congenital type of diverticulum found in the ileum. The edges of its mouth are well defined, sometimes being sharp and crescentic, as the diameter of the diverticulum beyond the opening into the bowel immediately begins to increase. The edge may be in continuity with one of the plica inside the lumen of the intestine. The lining of the pouch is smooth, and there is a complete peritoneal covering. In all the cases there is a close relation either to one of the ducts piercing the duodenal wall, or to a blood vessel.

When one holds the opened duodenum up to the light, the thinness of the diverticular wall compared with that of the duodenum is at once evident, and it is clear from this simple test alone that there is a gross deficiency of the muscular coat.

Microscopic examination of the wall of the diverticulum confirms this view. If the section is taken from the fundus, practically no muscle tissue is to be seen, except for a thin layer, not more than one or two fibers deep, usually to be found, and which represents the muscularis mucosæ. This layer, as in diverticula of the large bowel, may be well defined, being thicker, in fact, than in the intestinal wall. This is due to compensatory hypertrophy owing to the deficiency of the muscularis proper. As the wall is examined toward the mouth of the sac, muscle fibers may become more abundant. If a section is now examined which passes through the edge of the opening into the bowel, so that portion of the wall of both the diverticulum and the bowel be included, it is at once clear what has happened. At the angle formed by the abrupt change of direction of the lumen at the lip of the mouth, the muscular coat of the intestine appears to be unusually thick. Immediately, however, there is a diminution in thickness, shared by both the circular and longitudinal fibers, and both layers become equally and progressively attenuated until the muscular coat fades away completely, or is represented by an interrupted thin line of muscle not more than one or two fibers in thickness. Toward the fundus, it may be difficult to decide whether or not such fibers belong to the muscularis mucosæ.

The mucous membrane itself is thinned, but in uncomplicated cases shows little depar-

ture from normal, except in the absence of any Bruenner's glands.

The microscopic description given does not tally with that of all observers. For example. Baldwin states that all his 15 postmortem cases are "true," as they all contained muscle. But he says this consisted of a single layer, and therefore probably belonged to the muscularis mucosæ. In his summary of the literature previous to 1911, in 47 cases no microscopic report was made. In 20 other cases muscle was found, and in the 16 remaining it was stated to be absent. Jenkinson mentions an operation case with muscle in the wall but gives no detailed description. Basch in another operation case states "... muscular coat thin, but intact." Jackson describes a case in which the muscularis mucosæ is hypertrophied, with a definite but thin true muscle coat. In Moore's case, the muscularis mucosæ is thickened, but there is no true muscle coat. In Rolleston and Fenton's 4 cases, no muscle was found at all. Diverticula secondary to ulcer have a complete muscular coat. They are in no sense herniations of the mucous membrane through the muscularis of the intestinal wall, and a great deal of the conflicting evidence is due to confusion between the ulcer type and the primary type of diverticulum.

It may be concluded therefore that in primary diverticula of the duodenum the true muscular coat is grossly deficient, sometimes to the extent of complete absence.

AGE INCIDENCE

The average age in 25 of these cases is 51.9 years, the eldest being 73, and the youngest 31. In Cryderman's 45 cases diagnosed by X-ray, the average age was 51.7, the youngest being 24. There is thus a close agreement between the two series.

There is a striking difference, however, in the average age figure between these two X-ray series, and the postmortem series of Linsmayer, Baldwin, and Nagel, as can be seen in the tabulation on age incidence. If the average age of the combined postmortem figures be taken as 63, a difference of no less than 11 years exists—and is a striking indictment against the policy of accepting the age incidence of any conditions of this type which is based upon postmortem material alone. In 3 of the author's postmortem cases, the average age is 76.

AGE INCIDENCE

		Cases	Average age	Youngest	Oldest
X-ray	Author	26	51.9	31	73
	Cryderman	45	51.7	24	
Postmortem	Linsmayer	33 (single cases)	68	36	87
	Baldwin	15	60	47	82
	Nagel	20	61	33	87

It is difficult to determine the youngest recorded case of primary diverticulum, for records of the past have been so mixed up with diverticula associated with ulcer (in which the age incidence is younger) that one is forced to ignore them. Cryderman's youngest case of 24 might be accepted with the proviso that it was an X-ray diagnosis, and did not pass the absolute test of visual inspection. Huddy's case of acute perforation of a diverticulum of the second part was in a woman of 27. Pilcher's case, reported in 1894, of an enormous pouch interrupting the transverse portion of the duodenum in a boy of 17, had a wall of inflammatory fibrous tissue. It was in all probability a false diverticulum resulting from perforation of the duodenum. At all events, the pathology is not sufficiently clear to warrant its acceptance as a primary duodenal diverticulum.

The point which emerges quite clearly from this discussion is that primary diverticula of the acquired type are unknown before the age of 24, and rare before the age of 30.

SEX INCIDENCE

Of 26 cases, 15 were in women and 11 in men. The average age at discovery of the diverticula is, men 49 years and women 52.6 years. Linsmayer's figures are, women 27, men 14, and Cryderman's, women 30, men 14. In Baldwin's summary of 67 specimens found in the literature up to 1911, the sex was stated in 35 cases; 21 of which occurred in men, and 14 in women. This series cannot, however, be regarded as an accurate repre-

sentation, as certainly not all the cases included are of the primary type.

Of the 263 cases found in the literature in which the sex is mentioned, after excluding all cases in which there is any doubt as to the type of diverticulum described, the distribution between the sexes is: men, 112; women, 151.

SITUATION

By far the greatest number of primary acquired diverticula occur in the second part of the duodenum in close relation to the entrance of the common bile and pancreatic ducts.

The author has not been able to find a single proved case of primary diverticulum in the first part of the duodenum. This is an observation of the greatest significance, for although not ruling out all possibility of their occurring in this situation, it denotes at least that they, like true congenital diverticula, must be of extreme rarity, and this fact permits the clinician to regard all diverticula of the first part as secondary diverticula. The vast majority of these are due to the presence of an ulcer.

Diverticula of the third part of the duodenum, though far less frequently found than in the second part, are by no means uncommon. In the 26 X-ray plates, 3 postmortem cases and 1 operation case (the first postmortem specimen is included with the X-ray plates), the distribution is as follows:

First part		0
Second part		22
Third part*		9
Horizontal limb	4	
Vertical limb and flexure	5	
Total		31

*1 case with 2 diverticula

DISTRIBUTION

The more complete of the series to be found in the literature are shown in the tabulation on distribution.

If the author's cases are added, it will be seen that of the 280 cases in which the position has been noted, no less than 204, or 73 per cent, are situated in the second part.

These figures however, show considerable variations in the relative frequency in the various parts. Whereas Baldwin, and Spriggs and Marxer, find a steady diminution in frequency from the second part to the flexure, in the author's figures diverticula of the ascending part and the flexure and the horizontal limb are in the proportion 5 to 4. The most marked variation from the mean is in Linsmayer's records, for in his 41 cases examined post mortem, *all* of them were in the second part. One is at a loss to account for this, for anyone who studies Linsmayer's excellent article cannot fail to be convinced that his observations have been made with care.

DISTRIBUTION

	Year	Author	Total	First part	Second part	Horizontal part	Vertical part
Post-mortem	1911	Baldwin	15	0	9	5	1
	1914	Linsmayer	41	0	41	0	0
X-ray	1920	Case	68	0	49	19	
	1920	Cole and Roberts	26	0	23	2	1
	1925	Spriggs and Marxer	50	0	30	16	4
	1927	Cryderman	49	0	30	19	
			249	0	182	67	

MULTIPLE DIVERTICULA

In only 1 personal case was more than one diverticulum found. Two diverticula are not, however, uncommon, as reference to the literature shows. More than two are rare. In an unusual case described and fully investigated by Odgers no less than five pouches were present. Their distribution is as follows: the first, 4 centimeters above the ampulla of Vater; the second and third, perivaterien; the fourth, at the upper border of the transverse part, dorsal to the pancreas; the fifth, at the junction of transverse and ascending parts.

This specimen has been beautifully dissected and is to be found in the Museum of the Royal College of Surgeons.

In their X-ray survey, Spriggs and Marxer found two pouches in each of 38 patients. In 12 specimens, Keith found two pouches in 3 cases. These were paired on either side of the ampulla. Rolleston and Fenton describe a "bifid" diverticulum—identical, apparently, with Keith's in appearance—having a pouch on either side of the ampulla. These authors suggest that this was originally a single pouch

Fig. 1. Diverticulum of the duodenum associated with ulcer. (From a sketch made at operation.)

divided into two compartments as a result of tightening of the common bile duct following visceroptosis.

Fleischman describes a case with four diverticula, 1 with three, and a third with two, and Roth 2 cases with two diverticula in each.

In 20 cases Nagel found 6 with two pouches. All were perivaterien. Linsmayer in 41 cases found 7 with two, and 1 with three.

The diverticula are all necessarily related to the pancreas, lying either behind it, or partly buried in its substance. In the 15 cases of Baldwin, 8 were posterior, 3 lying below it, and 4 in its substance.

MORBID ANATOMY IN RELATION TO PATHOGENESIS

Situation in relation to the wall of the bowel. In all cases, without exception, the diverticula arise from the concave surface of the bowel, and are therefore closely related to the pancreas,—a relation which those seeking a congenital theory of origin emphasize very strongly. There is little doubt, however, that the situation on the concave surface is due to the weak areas in the bowel occasioned by the passage of the common bile and pancreatic ducts and the blood vessels. The greater size of the common bile and pancreatic ducts, and the long passage obliquely through the wall lead one to expect a greater frequency of primary diverticula associated with it than with the vessels, and indeed it is so, as the figures heretofore given show. The third part of the duodenum, though nearly twice as long as the second portion, provides only 27 per cent of the total number of cases. All these cases of the second part lie in the concavity of the duodenum, and at or near the ampulla of Vater. Although one cannot define the exact relation of the diverticula to the ampulla in cases examined by X-ray alone, postmortem records show that the greater number are intimately related to it. So common is this relation that French writers have coined the descriptive term "perivaterien" for diverticula of the second part of the duodenum.

Anatomy. The common bile duct and pancreatic duct of Wirsung run obliquely through the duodenal wall for one-half to three-fourths of an inch, and open into the ampulla of Vater on the left wall of the duodenum slightly to the posterior aspect. The union of these ducts does not take place until they have almost reached the opening in the duodenal papilla. The exact mode of termination of the ducts is subject to slight variations.

The passage of the ducts through the duodenal wall necessitates a considerable gap in the muscle fibers. The longitudinal coat is most affected, for in the angle between the tracks of the pancreatic and bile ducts, a well defined circular coat exists, whereas the divarication of the muscle fibers of the longitudinal coat is practically continuous.

The pancreatic duct of Santorini pierces the duodenal wall three-fourths inch above and somewhat anterior to the main duct, and terminates in a minute orifice which is often difficult to find even on examination of postmortem specimens.

The weakness occasioned by the separation of longitudinal fibers probably continues for some distance below the entrance of the ducts.

The blood vessels. The duodenum is supplied by the inferior pancreaticoduodenal artery of the superior mesenteric and the superior pancreaticoduodenal of the gastroduodenal branch of the gastric artery. The first part also receives some branches from the pyloric artery.

Figure 9 illustrates the dissection of blood vessels after injection with carmine. For the

sake of clearness, the pancreas has been dissected free from the superior mesenteric vessels and common bile duct. This has necessitated division of the small branches of the pancreaticoduodenal arteries supplying the pancreas, but has left the duodenal branches undisturbed.

The inferior pancreaticoduodenal is a short thick trunk at its origin from the superior mesenteric, and almost immediately gives rise to a large branch which passes to the left and follows the concave curvature of the duodenum (under cover of the inferior margin of the pancreas) to the flexure. In the specimen dissected the main artery now divides into two branches, from which the bowel is supplied by a double arcade, anterior and posterior. The anterior branches are easily visible under the peritoneum covering the anterior wall of the duodenum, but the posterior branches run under the lower margin of the pancreas.

As the vessels pass to the right they run to either side of the common bile duct, and terminate by anastomosing with similar dorsal and ventral branches of the superior pancreaticoduodenal artery. The dorsal and ventral branches in this case arise independently from the gastroduodenal artery.

This arrangement of the duodenal blood supply does not correspond in detail with that described in standard textbooks of anatomy. Similar findings, however, are reported by Yule, whose article the author discovered after his own dissections were complete.

There is thus a double arcade of supply to that part of the duodenum from a point above the entrance of the common bile and pancreatic ducts and the point at which the superior mesenteric artery crosses the intestine.

If a cross section of the horizontal portion of the duodenum be examined, it is seen that the anterior and posterior series of branches are separated by a considerable interval—five-eighths to three-fourths inch at the points at which they pierce the duodenal wall (Fig. 10).

This relation coincides with the situation of diverticula in the third part. Some of the latter are easily visible, and anterior, their opening being in the same plane as the anterior branches. Others are posterior, and hidden, the opening corresponding with the posterior branches.

Fig. 2. Pulsion diverticulum of the second part of the duodenum; 1¼ hours after a barium meal. The barium in the colon is due to a barium enema given 2 days previously.

Perivaterien diverticula. Perivaterien diverticula are situated in the concavity of the duodenum, immediately above the opening of the common bile duct.

In Keith's article, 7 single cases are described in this situation. In the 3 cases in which two diverticula are present, the pouches appear one on either side of the common bile duct. The right one is in each case the larger. One cannot fail to appreciate Keith's view that in the cases in which two are present we are seeing merely a further stage in the single cases in the formation of these diverticula.

A few cases are to be found in the literature in which the common bile duct or pancreatic duct, or both, actually open into a diverticulum. I suspect some, at least, of these cases as being exaggerations of the condition of wide mouthed opening of the common bile duct. Traction by the common bile duct following inflammation in the biliary tract might cause such an opening to be pulled up into a

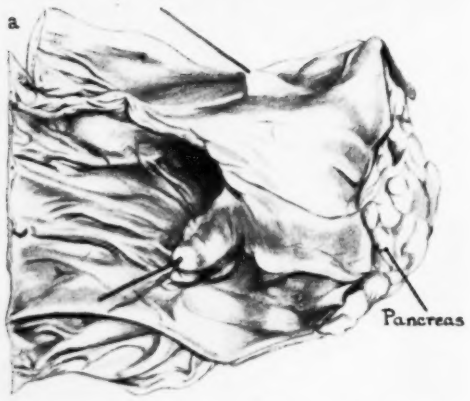


Fig. 3. Diverticulum of the second part of the duodenum.

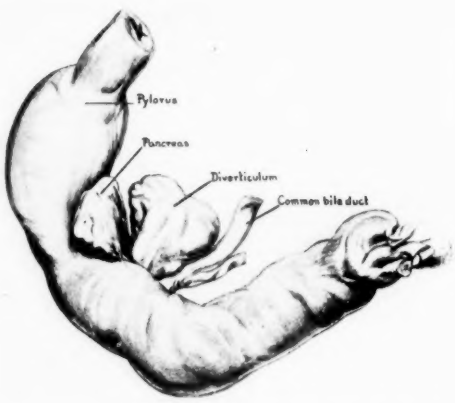


Fig. 4. Diverticulum of the second part of the duodenum.

diverticulum of considerable size, as in the case recorded by Benninghoven and Michael. One case in which the accessory duct of the pancreas opened into a diverticulum is also recorded.

The second most frequent site in the second part of the duodenum appears to be above the ampulla, but in the same plane, and associated with the opening of the accessory pancreatic duct of Santorini in the minor papilla.

Baldwin's analysis of the cases in the second part in the literature up to 1911, and including his own, is as follows:

	Cases
Convexity of duodenum	0
Concavity of duodenum	58
Near pylorus	5
Near minor papilla	9
With accessory duct opening into it	1
Near major papilla	12
At major papilla	24
Wirsung's duct opening into it	3
Both ducts opening into it	4

THE THIRD PART—TRANSVERSE, ASCENDING AND FLEXURE

In the author's case of double diverticulum, the relation between the mouths of the diverticula and the entry through the intestinal wall of two posterior branches of the superior pancreaticoduodenal artery is clearly shown. This relation is so close that there can be no doubt that the pouches herniated through the gap in the musculature occasioned by the entry of the blood vessels. Grant describes a similar case in which were present two diverticula of the transverse part in the line of the opening of the common bile duct, suggesting that a weakness persisted in the longitudinal muscle fibers after being pierced by the duct for some distance along the concave border of the duodenum. That this may be a factor cannot be disputed. A large vessel was, however, associated with both diverticula. It bifurcated at the fundus, one branch going to the posterior and the other to the anterior surface of the diverticulum in each case.

When the diverticulum, which commences as a small herniation of the mucosa through a gap in the muscular wall, increases in size as a result of intraduodenal pressure, its mouth increases in size, and takes up more and more of the diameter of the intestine. Thus, in a number of cases, the exact point of commencement of the pouch cannot be identified. A protrusion of the mucosa occurs on either side of the midline, corresponding with the entry of a vessel. As these increase in size, they eventually fuse, opening into the bowel by a wide mouth which bestrides the midline (corresponding with the mesenteric attachment in the case of the jejunum).

The ease with which diverticula are found at operation will depend largely on whether they start in association with the anterior or posterior branches of the superior pancreaticoduodenal artery. The latter will be completely

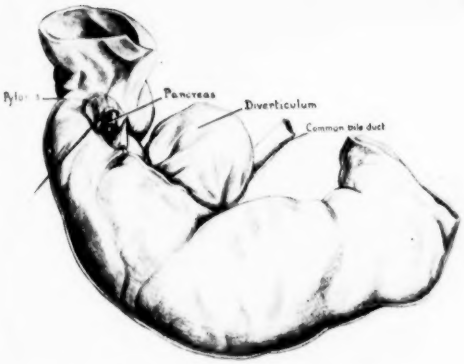


Fig. 5. Diverticulum of the duodenum.

obscured by the overlying pancreas. In the transverse part the mouths of the diverticula open downward, and the pouches therefore drain readily. The diverticula, probably because of this, do not tend to get large. The fact that only 2 cases were shown in the author's series of 26 X-ray cases may be accounted for by the same token, the barium only remaining in the diverticulum for so short a period of time that it escapes the eye of the examiner.

The terminal portion and flexure. Diverticula of the flexure pass inward to the right, and below the lower border of the body of the pancreas. This situation is constant, though the exact point in the diameter of the bowel from which they arise has not been determined.

Their semidependent position may account for the large size to which they sometimes develop. Jacquelin and Quénu report a diverticulum, removed at operation from a woman of 54, which opened from the bowel near the flexure, and passed to the right behind the peritoneum as far as the left border of the duodenum. It measured 8 by 2 centimeters and was easily seen in X-ray examination. Lund describes a very similar case in which operation was also done. The diverticulum passed from the region of the flexure behind the first part of the duodenum to reach the posterior aspect of the gall bladder. X-rays had shown its presence before operation. One or two diverticula were also found in the beginning of the jejunum.

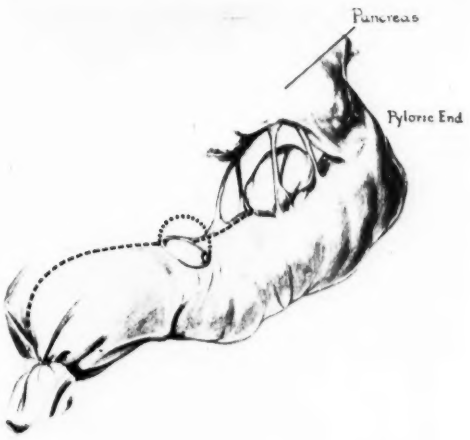


Fig. 6. Diverticula of the third part of the duodenum.

Diverticula at the flexure play a prominent rôle clinically owing to their tendency to cause symptoms suggesting obstruction to the passage from the duodenum into the jejunum. Krogius describes a case of acute obstruction due to this cause in a woman of 64.

THE PATHOGENESIS

That diverticula such as have been described in the preceding pages are acquired and not inborn is placed beyond reasonable doubt by the age incidence of their discovery. One cannot on the same ground, however, dispose of the possibility of a congenital factor being responsible, at all events in part, for their development. Though I should like to state at once that my own investigations do not lead me to subscribe to the likelihood—or even the necessity for postulating such a factor—one cannot pass by the views of men such as Professor Wilkie, who formulated so emphatically a congenital theory of origin some 20 years ago, without being conscious of some loss of confidence in the validity of one's own conclusions.

The first source of guidance as to the pathogenesis lies in the structure of the wall of these diverticula. There is uniformity of opinion that the muscular coat is deficient. In all my own cases there was a complete lack of muscle at the fundus of the diverticulum, except

Fig. 7. Diverticulum removed at operation from the second part of the duodenum.

Fig. 8. X-ray appearance of diverticulum shown in Figure 7, 24 hours after commencement of a barium meal.

for that belonging to the muscularis mucosæ. Failure to state the exact place in the diverticulum from which a section was cut, and upon which the opinion was formed as to the nature and quantity of muscle found, compels one to rely on one's own limited pathological investigation and the comparatively few cases in the literature in which this is clearly stated.

Two views are possible with regard to the muscular deficiency. Either the muscle is deficient from the commencement, or it was normal to begin with and became thin as a result of pressure. This latter event appears to be the explanation of most of those who regard the diverticula as of congenital origin. This sequence of events is, however, unlikely. Congenital diverticula of the ileum do not behave in this way, and indeed there is no reason why abnormal pressure inside the duodenal lumen should of its own accord cause atrophy of the muscle of the diverticular wall any more than that of the duodenum whence the diverticulum opens. Tandler has shown that in the early stages of development of the duodenum the epithelial lining of the gut develops in advance of the mesodermal coats, and Wilkie suggests that the outgrowth of an accessory pancreatic bud in the region of the hepatic and the two pancreatic ones at such a stage might result in a diverticulum with an imperfect mesodermal sheath. But in not one of the author's cases was there any question of direct connection between the pancreas and the diverticulum. The latter was in each case separated by a layer of areolar tissue from the pancreas, and there was no difficulty in dissecting it free.

The lack of evidence is quite clearly in favor of the view that these diverticula are hernial protrusions of the bowel lining beginning at a weak spot in the muscularis. The point is being labored somewhat, for it is the key to the pathogenesis, and unless one is fully prepared to accept this view what follows is not tenable.

The formation of any type of hernia, depends upon the interworking of two factors: (a) an area of diminished resistance in the wall enclosing the cavity from which the hernia arises and (b) pressure inside this cavity.

Areas of diminished resistance are furnished in the duodenum by the piercing of its muscular wall by the bile and pancreatic ducts and by the blood vessels, and the constancy with which diverticula are associated with one or other of these structures is sufficient

Fig. 9. The vascular supply of the duodenum. A dissection made after injecting the arteries with carmine jelly.

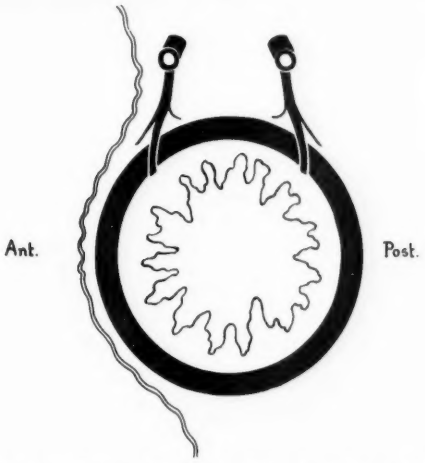


Fig. 10. The vascular supply of the duodenum. A diagram to illustrate the relation to the bowel wall of the sites of entry of the blood vessels.

proof of the contention. The larger the structure piercing the wall, the more frequent should be the presence of a hernial pouch, and the facts of incidence bear this out quite closely.

DUODENAL PRESSURE

The pressure inside the duodenum depends upon (a) the contents and (b) the contraction of the muscular wall.

What is the normal pressure in the duodenum? The musculature is as powerful as that elsewhere in the small intestine, therefore a high pressure during contraction is conceivable. But, if the pylorus is patent, the duodenum is open at both ends, and under normal conditions of segmental and peristaltic movements, the pressure is not greatly raised.

The duodenal pressure has been examined in 13 healthy patients. A radio opaque tube is swallowed by the subject until it is seen under the X-ray screen to be past the pylorus. It is then connected to an ordinary water manometer, and the water levels in the two limbs compared. In all cases there was practically no difference caused in the levels, except a rise and fall of from 0.5 to 1 centimeter corresponding with the respiratory excursion of the diaphragm.

If a reading could be made in this way with the pylorus closed, and the duodenum contracting, it is probable that greater differences in the pressure reading would result. The rapidity with which the stomach contents pass through the duodenum, as demonstrated by X-ray, does not suggest that under normal conditions the duodenal contents exert any appreciable pressure upon the duodenal walls.

That under abnormal conditions, at least, a high pressure may occur in the duodenum is shown by the case which Figure 11 illustrates.

This remarkable appearance was discovered during the course of a barium meal in a female patient aged 62. It shows the common bile duct, the left and right hepatic ducts and even the smaller intrahepatic canaliculi filled with barium. The commencement of the cystic duct is shown, but not the gall bladder. This appearance was present in the roentgenogram taken immediately on swallowing the meal, and remained until the 3 hour picture was taken. At 24 hours, the smaller canaliculi could still be seen, but the larger ducts were empty. At 48 hours, the liver was clear.

This case furnishes unquestionable evidence that under abnormal conditions at least, the pressure inside the duodenum may

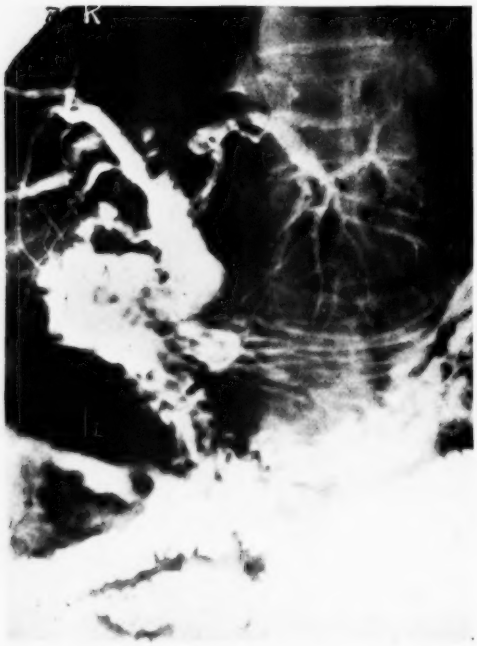


Fig. 11. Duodenal pressure. Entry of barium into the hepatic ducts during a barium meal.

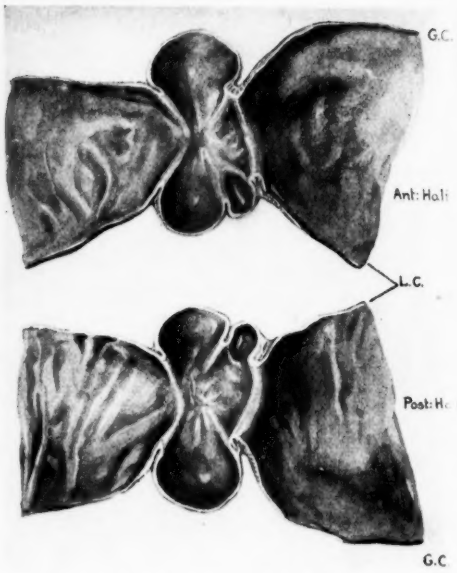


Fig. 12. The ulcer diverticula of the duodenum.

rise to a very considerable height. Further evidence that high duodenal pressure occurs lies in the formation of ulcer diverticula, for there is no question that intraduodenal pressure plays a part in the production of these. As Perry and Shaw have pointed out, the duodenum is the only situation in the alimentary tract in which ulcers are followed by diverticula. These workers think the pylorus is largely responsible for a rise in intraduodenal pressure in opposing, by its contraction, an obstacle to the reflux when the lower part of the duodenum is contracting. The pressure here is greater than elsewhere in the small intestine, where the fluid constituents are distributed over a greater length of bowel. In support of this theory of formation of diverticula immediately *distal* to an obstruction, these authors describe a case of diverticulosis of the jejunum associated with a partial congenital diaphragm situated at the duodenojejunal flexure in a man of 40. The aperture in the diaphragm admitted the fifth finger, and there were three diverticula measuring 1 by 1 inch on the mesenteric border of the first foot of the jejunum.

How is this high pressure produced? Not by normal segmental and peristaltic contractions, but by irregular inco-ordinated tonic contractions of the circular and longitudinal coats.

Given, therefore, weak areas in the bowel wall, and the possibility of high pressure being caused inside the lumen of the duodenum by irregular muscular contraction, an explanation of the mode of origin of these primary diverticula becomes possible.

The mucous membrane of the duodenum is forced into the interval occasioned by the passage through the duodenal wall either of the ducts or the blood vessels by the circular contractions of the duodenal muscle. In the early stages, the mucous membrane will recover its position, but once started there will be a certainty of recurrence, and a repetition of the event over a number of months or even years will result in a stretching of the interval between the muscle fibers, and its permanent occupation by a wedge-shaped pouch of mucous membrane. This process is easier to study both in the jejunum and the

colon, where the opportunity for observing the earliest stages arises. There can be no doubt, judging from the pathological anatomy, that this is the way that all primary pulsion diverticula start irrespective of their situation in the alimentary tract.

Once the process of mucous membrane in an intermuscular interval is established, the formation of a typical pouch is inevitable, if the patient lives long enough. The resistance to the pressure inside the duodenum is at a minimum. As the pouch is forming, it carries with it muscle fibers from the bowel, the quantity varying in different cases, but consisting of a very thin layer, or being entirely absent at the fundus of the diverticulum, which was the first portion to herniate. The subsequent size to which the pouch develops depends upon its age—the very large ones are nearly always found in old people—and its position. For example, those in the transverse portion of the duodenum are usually small, because of the absence of any stretching force due to the weight of food contents. Those in the second part of the duodenum are probably limited in size by the support given by the pancreas, but those connected with the flexure may become relatively enormous.

In the two latter situations, there is practically always a certain degree of stasis as shown by the barium meal and X-ray. The failure to empty synchronously with the duodenum is due at first to the lack of an adequate muscular coat.

The high pressure to which the herniation is due is undoubtedly intermittent, and must be continued over a long period of time. Diverticula do not arise as a result of the presence of an acute obstruction to any part of the bowel.

There is an apparent paradox in this theory of development of pulsion diverticula. Both the degree of pressure and the strength of the duodenal wall depend upon the development of the muscular coat. Therefore duodena in which the muscular layer is best developed would have the most impervious wall. But the difficulty passes when it is realized that the muscular contraction which produces pulsion diverticula is an irregular one, there being areas of relaxation of the muscle coat intensified between the sites of tonic contraction.

The question of relaxation is an important one. If one segment of muscle is prone to enter into long periods of overaction, it should tend to hypertrophy, or at least to maintain its power. What changes may take place in a neighboring segment, which is not only relaxed for similar periods of time, but has exerted upon it during its relaxation an abnormal pressure generated by the contracting portion? Does it undergo a "relaxation atrophy"? That such a change is associated with diverticula of the large intestine, which are morphologically identical with those of the small intestine, will be made clear from a study of specimens. The relaxation area in these gives a mirror image picture of the neighboring contraction area. Such a relaxation atrophy, should it occur, as I believe it does, would explain the ease of formation and progressive increase in size of the diverticula.

The presence of rugæ in some of these diverticula is not inconsistent with the theory of pulsion, for the causal pressure is an intermittent one, and Grey Turner's case of duodenal diverticulum in a woman of 60 in which the mucous membrane was thrown into folds, is not necessarily contrary evidence.

THE RELATION BETWEEN DIVERTICULA AND VISCEROPTOSIS

In many cases X-rays show the presence of varying degrees of visceroptosis in subjects with duodenal diverticula, and it has been suggested that in this condition the vessels and bile ducts cause traction upon the intestinal wall, and tend to pull up the mucous membrane through the muscle coat. There may be some truth in this theory with regard to the formation of diverticula in the jejunum, but it is improbable that the duodenum reacts in this way, as its comparative fixity to the posterior abdominal wall protects it from sharing in the visceroptotic process except in the severest cases.

The view that mucosal hernias are more likely to occur in the subjects of visceroptosis because of the "atonicity" and consequent weakness of the bowel wall found in this condition can be entirely discounted. There is

abundant evidence to show that abdominal ptosis bears no relation to the condition of the muscular wall of the hollow viscera.

A possible predisposing cause of increased pressure in the duodenum occasioned by visceroptosis is constriction of the transverse portion by the superior mesenteric vessels. That this is not of very great importance, though possibly a contributory factor in some cases, is shown by lack of association between ileus of the duodenum and diverticulosis. This lack of cause and effect between chronic ileus and diverticulosis may appear to be evidence against the pulsion theory. This is not the case, however. The facts merely show that diverticula do not arise solely as a result of passive pressure of contents upon the intestinal wall.

ACQUIRED DEGENERATION OF THE MUSCLE COAT

Is a primary local degeneration of the muscular wall a factor in producing a locus minoris resistentiæ? Inflammation of the muscle coat leading to atrophy has been cited as a cause, but there is no histological evidence of this. Further, the constant situation in the concavity of the duodenum would not be found if such were the case. Senile atrophy is another hypothesis, but one which is readily discounted. Emphatically, duodenal diverticulosis is not a disease of old age, but of early middle age. It is true that postmortem evidence suggests its greater frequency in the aged, but the fallacy of this has already been pointed out. It is true, too, that generally speaking, the older the subject the larger the diverticulum. But this is to be expected. The diverticulum tends always to get larger, and the older it is the larger it will become.

THE ULCER DIVERTICULUM

Ulcer diverticula form a distinct group. In contradistinction to primary diverticula, the ulcer diverticulum is found more readily at operation or at postmortem than by X-ray. At postmortem examination, the one thing that may easily be missed is not the diverticulum, but the healed ulcer which was responsible for it. All that is to be seen, in most cases, is a minute smooth whitened area of mucous membrane adjacent to the diverticulum. The opened duodenum must be examined in a good light, if necessary with a lens. The smallness of the visible ulcer is well shown in the accompanying illustration made from a postmortem specimen. In this the artist has, for the sake of clear definition, purposely accentuated the appearance of the ulcer scar. It will be appreciated, therefore, how easily such scars may be missed at postmortem examination.

The only other type of diverticulum which occurs in the first part of the duodenum is the true congenital type, and this is so excessively rare that one can safely work on the assumption that if a diverticulum is found in the first part of the duodenum, there too will the scar of an ulcer be found.

Morbid anatomy. Both the diverticula and the ulcers may be multiple. In 7 postmortem specimens, the numbers were as follows:

1. 2 cases with 1 ulcer and 1 diverticulum.
2. 1 case with 2 ulcers and 1 diverticulum.
3. 2 cases with 2 ulcers and 2 diverticula.
4. 1 case with 2 ulcers and 3 diverticula.
5. 1 case with 2 ulcers and 4 diverticula.

Each diverticulum consists of all four layers of the normal duodenum, and the peritoneal coat is free and smooth. The mouth is well defined and bounded by a definite ridge of mucous membrane, and the lining of the pouch is smooth. There is no inflammatory process in its wall, and the associated ulcer lies at some distance from its edge, and not in the floor or wall. The following is selected as a typical example:

Specimen 6 (*Fig. 12*). The pyloric end of the stomach and the duodenum have been divided into halves. The lesser curvatures lie next to one another in the illustration.

One-half to three-quarters of an inch distal to the pylorus are two scars of healed ulcers, occupying the anterior and posterior walls of the duodenum, respectively. Just distal to the ulcers is a prominent fold consisting of the whole thickness of the wall encircling the duodenum. Between this fold and the pylorus, the duodenum is pouched out. The pouch on the lesser curvature is subdivided by a well marked fold, again consisting of the whole thickness of the duodenal wall, into two compartments. The mucous membrane lining the pouches is smooth, and their walls are somewhat thinner than that of the rest of the duodenum, but there has been no interference with the muscle coat.

Pathogenesis. The mode of formation of these diverticula is well illustrated by this specimen. The duodenum has been shortened by contraction of scar tissue of the healed ulcers, so that the wall of the duodenum unoccupied by the ulcers has been puckered up, and ballooned outward by intraduodenal pressure to form diverticula. In this case, the maximum contraction has taken place in the direction of the long axis of the bowel.

The diverticula are produced by contraction of scar tissue, and not by yielding of a weak area in the wall caused by ulceration. The latter probably never occurs. The dense fibrosis produced by the healing of a peptic ulcer would be the last place for the duodenal wall to give if subjected to pressure. In this view, the author is at variance with those expressed in the literature, in particular by Thorek and F. C. Turner, both of whom describe diverticula caused by yielding of an ulcer, and describe cases in which the floor is formed by an ulcer crater. Such cases are to be regarded not as diverticula but as a part of the process of destruction of a large penetrating ulcer. A third type is described in which an ulcer is associated with a pulsion diverticulum of the second part. The part, if any, played by the ulcer in the formation of a perivaterien diverticulum is little understood, and therefore the association of the two is best regarded as accidental.

The amount of shortening of the duodenum caused by the contraction of scar tissue is considerable, especially in the specimen described, where the unaffected portion of the duodenum is so ballooned out. In this case, the shortening by actual measurement is 3.6 centimeters on the greater curvature side.

That pressure plays a part, albeit a subsidiary one, in the production of these diverticula is shown by the lack of rugæ on the floor in nearly all the cases. In no case is there any gross deficiency of the muscular coat. The slight difference in thickness shown in some cases between the diverticula and the duodenal wall is due to attrition following the prolonged effect of intraduodenal pressure. But another explanation is possible. The normal contracting power of the duodenum is impeded by the fixed deformity due to the ulcer, and there may therefore be a disuse atrophy of the muscular wall in the region of the deformity. It is probable that the persistent spasm of muscle associated with ulcer plays a part by helping to create the deformities, which are subsequently fixed by the scar contraction.

Whatever be the true nature of this minor degree of thinning of the muscle coat, the point which the author wishes to make quite clear is that the diverticula are due primarily to puckering of the duodenum due to scar tissues, and the effect of intraduodenal pressure is of secondary importance.

These diverticula differ therefore in situation, morphology, and pathogenesis from the primary pulsion type met with in the second and third parts of the duodenum.

TRACTION DIVERTICULA

In the series of 31 cases of duodenal diverticula diagnosed by X-ray, there are 2 examples of diverticula caused by the contraction of scar tissue adherent to the wall of the duodenum. In one of these the diverticulum is situated near the flexure, and is caused by involvement of the duodenal wall in the healing process of an ulcer of the lesser curve.

The second case occurred in a woman patient of 73 and was confirmed at operation. The gall bladder was thick and fibrotic, and densely adherent to the junction of the first and second parts of the duodenum, the wall of which was pulled out by the adherent gall bladder to form a small pouch.

Perry and Shaw describe a similar case. In a patient with chronic cholecystitis the wall of the duodenum was adherent to the liver, and drawn by adhesions into the form of a narrow pointed cul-de-sac. In a second case of these authors, a gall stone was found in a pouch on the right wall of the duodenum midway between the pylorus and the papilla. The pouch was adherent to a shrunken and fibrosed gall bladder. The case suggests the formation of a traction diverticulum, with later a cholecystoduodenal fistula which allowed the gall stone to enter the pouch, the opening healing after this had taken place.

That traction upon the common bile duct might pull up the wall of the duodenum into

a pouch is illustrated by the case of Benninghoven and Michael. At postmortem in a man of 53 with a history of many years of epigastric pain, the common bile duct was dilated and had a very thick wall. It opened into a diverticulum measuring 2½ centimeters in diameter. The gall bladder was obstructed by a fibrous band across the cystic duct, and there were dense adhesions around the duodenum. The diverticulum contained all four layers of the normal bowel.

PSEUDO-DIVERTICULA AND UNCLASSIFIED CASES

Perforation of the duodenal wall into the peritoneal cavity may give rise to a diverticulum, the walls of which are formed by fibrous tissue. Pilcher's case, which was previously mentioned, was undoubtedly of this type, to which the title of *pseudo-diverticulum* may be given.

Communication between the duodenum and the gall bladder may also be regarded as a pseudo-diverticulum. Korchow's case, in which a fistula formed between the gall bladder and the first part of the duodenum is an unusual example of this.

It is difficult to classify a few cases of duodenal diverticula recorded in the literature, mainly because insufficient data is furnished by the authors. Perry and Shaw mention a pouch of the posterior wall of the duodenum three-fourths inch from the pylorus, which communicated with the colon. Hunt and Herbst describe a remarkable case of duodenal diverticulum of large size, and full of gall stones which communicated by a fistulous track with the gall bladder. It is possible that it commenced as a primary pulsion diverticulum which subsequently became involved by the gall bladder, but the situation of the opening between the diverticulum and the duodenum does not conform strictly to the pulsion type. A more likely explanation is that of distention of a fistulous communication between the duodenum and the gall bladder.

DIVERTICULUM ASSOCIATED WITH GROWTH

There is one example of a duodenal diverticulum associated with malignant disease in the literature, reported by Morrison and Feldman.

At operation upon a man of 59, a tubular growth 1 by 2½ inches was found, arising from the cap of the duodenum, from which clear fluid was aspirated. At the mouth of the diverticulum was found an indurated mass the size of a walnut, and there were secondary nodules in the liver. No pathological investigation was made, so that one is in doubt as to the exact nature of the diverticulum and its relation to the associated growth.

My thanks are due to the Council of the Royal College of Surgeons for permission to publish this section of the Jacksonian Prize Essay of 1932, and I wish to acknowledge also my thanks to my colleagues of King's College Hospital, to Miss Barclay Smith and Mr. S. Steward for the illustrations and to Professor Stewart for the specimen illustrated in Figure 12.

BIBLIOGRAPHY

1. Anderson, J. H. Brit. J. Surg., 1922–1923, 10: 316.
2. Andrews, E. W. J. Am. M. Ass., 1921, 77: 1309.
3. Bainbridge, W. Texas State M. J., 1927, 22: 560.
4. Baldwin, W. M. Anat. Rec., 1911, 5: 121.
5. Basch, S. Am. J. M. Sc., 1917, 153: 833.
6. Bassett, V. H. Tr. Chicago Path. Soc., 1907, 7: 83.
7. Bauer, T. Wien. klin. Wchnschr., 1912, 25: 879.
8. Bell, H. H., Anat. Rec., 1921, 21: 229.
9. Bengolea, A. J. Bull. Soc. nat. chir., 1928, 54: 726.
10. Benninghoven and Michael. Radiology, 1931, 17: 579.
11. Bonneau, R. Presse méd., 1923, 31: 817.
12. Buckstein, J. Am. J. Surg., 1927, 3: 340.
13. Butler and Ritvo. Boston M. J., 1925, 192: 705.
14. Calder. Medical Essays and Observations, Edin., 1733, 1: 205.
15. Caraven, J. Presse méd., 1923, 31: 1084.
16. Carson, H. W. Tr. Med. Soc. Lond. 1930, 53: 76.
17. Case, J. T. Am. J. Roentgenol., 1916, 3: 314.
18. Idem. J. Am. M. Ass., 1920, 75: 1463.
19. Chlumsky, V. Beitr. z. klin. Chir., 1899, 25: 539.
20. Chomel. Hist. Acad. roy., Par., 1710, p. 37.
21. Cole and Roberts. Surg., Gynec. & Obst., 1920, 31: 376.
22. Cullen, E. K. Arch. Surg., 1921, 2: 542.
23. Davis, N. S. Tr. Chicago Path. Soc., 1913, 9: 1.
24. Delannoy and Driessens. Écho méd. du nord., 1931, 35: 301.
25. Downes, W. A. Ann. Surg., 1922, 76: 43.
26. Erdmann, J. F. (Ref.) Ann. Surg., 1922, 76: 43.
27. Fairland, E. Brit. M. J., 1879, 1: 851.
28. Falconer, A. W. Lancet, 1907, 1: 1296.
29. Fischer, M. S. J. Exper. Med., 1900, 5: 333.
30. Fleischman. Leichen Offnungen, 1815, p. 1.
31. Forsell and Key. Nord. méd. Arch., 1915, 2: 48.
32. Gask, G. E. St. Bart's Hosp. Rep., 1929, 62: 122.
33. Gillet. Arch. de mal. de l'appar. digest., 1930, 20: 212.
34. Gordinier and Sampson. J. Am. M. Ass., 1906, 46: 1585.
35. Grant, J. C. B. J. Anat., 1923, 57: 357.
36. Grasberger, R. Wien. klin. Wchnschr., 1897, 10: 149.

37. Habersohn. Observations on Diseases of the Alimentary Canal, Lond., 1857, 145.
38. Hahn, O. Review of Literature. Ergebn. d. Chir. u. Orthop., 1930, 53: 76.
39. Harley. Tr. Path. Soc. Lond., 1857, 8: 235.
40. Hartung. Radiology, 1925, 4: 308.
41. Heacock, C. H. South. M. J., 1929, 22: 348.
42. Huddy, G. P. B. Lancet, 1923, 2: 327.
43. Hunt and Herbst. Surg. Clin. N. America, 1925, 5: 807.
44. Hurst, A. F. Guy's Hosp. Rep., 1929, 79: 420.
45. Jackson, C. M. J. Anat., 1908, 42: 219.
46. Jacquelin and Quénu. Bull. et mém. Soc. nat. de chir., 1927, 53: 716.
47. Kaldor, J. Ann. Surg., 1929, 89: 6.
48. Kampmeier, R. H. Am. J. M. Sc., 1926, 172: 590.
49. Keith, Sir Arthur. Brit. M. J., 1910, 1: 376.
50. Korchow, V. Zentralbl. f. Chir., 1928, 55: 2955.
51. Krogius, A. Deutsche Ztschr. f. Chir., 1927, 200: 273.
52. Larimore and Graham. Surg., Gynec. & Obst., 1927, 45: 257.
53. Lefevre and Jonchières. J. de méd. de Bordeaux, 1926, 103: 648.
54. Lewis, D. J. Am. M. Ass., 1921, 76: 783.
55. Linsmayer, H. Verhandl. d. deutsch. path. Gesellsch., 1914, 17: 445.
56. Lockhart, R. H. Yale J. Biol. Med., 1931, 3: 505.
57. Lockwood, A. L. Transactions of the Active and X-Resident Physicians of the Mayo Clinic, 1930, 10: 54.
58. Lund, F. B. New England M. J., 1928, 198: 986.
59. Mackechnie, H. N. Ann. Surg., 1921, 74: 96.
60. Maclean, M. J. Ann. Surg., 1927, 85: 73.
61. Idem. Surg., Gynec. & Obst., 1923, 37: 6.
62. McQuay, R. W. Ann. Surg., 1929, 89: 36.
63. Monsarrat, K. W. Brit. J. Surg., 1926, 14: 179.
64. Moore, E. C. Arch. Surg., 1920, 1: 513.
65. Moore, N. Tr. Path. Soc. Lond., 1884, 35: 202.
66. Morgagni. Seats and Causes of Disease, London, 1769, Book 2, Letter 34, Art. 17; Letter 37, Art. 25.
67. Morrison and Feldman. Ann. Clin. Med., 1925, 4: 403.
68. Moynihan, B. G. A. Duodenal Ulcer, London, 1912, 268.
69. Murchison, D. R. J. Am. M. Ass., 1920, 75: 1329.
70. Nagel, G. W. Arch. Surg., 1925, 11: 529.
71. Odgers, P. N. B. Brit. J. Surg., 1930, 17: 592.
72. Pendergrass, R. C. Am. J. Surg., 1928, 5: 491.
73. Penhallow. J. Am. M. Ass., 1923, 80: 1372.
74. Perry and Shaw. Guy's Hosp. Rep., 1893, 50: 171.
75. Pilcher, L. S. Ann. Surg., 1894, 2: 62.
76. Ritchie and McWhorter. Surg., Gynec. & Obst., 1917, 25: 485.
77. Rolleston and Fenton. J. Anat. & Physiol., 1900, 35: 110.
78. Roth, M. Arch. f. path. Anat., 1872, 56: 179.
79. Scott, S. G. Brit. M. J., 1931, 1: 346.
80. Spriggs and Marxer. Brit. J. Surg., 1920, 8: 2.
81. Ibid. 1921, 8: 18.
82. Idem. Quart. J. Med., 1925, 19: 1.
83. Tandler. Morphol. Jahrb., Leipzig, 1902, 29: 187.
84. Thorek, M. Illinois M. J., 1929, 55: 64.
85. Transactions. Section Rad. Am. M. Ass., 1927, p. 208.
86. Turner, F. C. Tr. Path. Soc. Lond., 1884, 35: 200.
87. Turner, G. Grey. Lancet, 1920, 1: 151.
88. Vorhaus, M. G. J. Am. M. Ass., 1930, 94: 165.
89. Weiss, K. Frankfurt. Ztschr. f. Path., 1929, 89: 6.
90. Wilkie, D. P. D. Edinburgh M. J., 1913, 11: 219.
91. Yule, E. J. Anat., 1926–1927, 61: 344.

THE MECHANISM BY WHICH THE ACIDITY OF AN ACID MEAL IS REDUCED IN THE STOMACH

FREDERICK C. HILL, M.D., LEO C. HENRICH, AND CHARLES M. WILHELMJ, M.D.,[1] OMAHA, NEBRASKA

THE "acid meal," since its introduction by Boldyreff, has been used rather extensively in experimental studies on the stomach, and has also been employed by several observers in clinical investigations. It consists of from 100 to 300 cubic centimeters of 0.3 per cent to 0.5 per cent hydrochloric acid, and may be administered either by mouth or, more commonly, by means of a stomach tube. This type of meal is emptied out of the stomach somewhat more slowly than water, and the stronger the acid is, the more the emptying time is prolonged.

When an acid meal of this character is introduced into the stomach and withdrawn 15 minutes to one-half hour later it is found that the acid, during its sojourn in the stomach, has been reduced in strength. This reduction is considered by most observers to be due to neutralization of the acid by: (1) mucus secreted by the stomach; (2) regurgitated fluid from the duodenum, consisting of bile, pancreatic juice and succus entericus. With reference to the first suggested cause of the reduction, the gastric mucus, we (12) have shown by studies on pouches prepared from the entire pyloric region of the stomach of dogs, and also by unpublished investigations on pouches which we prepared from the entire stomach, that the amount of gastric mucus available is not sufficient in quantity nor alkaline enough to account for any significant amount of the reduction noted. The most important factor concerned in reducing the acidity of an acid meal is thus not intragastric, but extragastric; it is the fluid which comes from the duodenum into the stomach.

That this regurgitation of duodenal contents is a common occurrence both in man and in experimental animals has been noted by various observers for many years. Thus in 1909 Ehrmann and Lederer noted considerable regurgitation in the Ewald-Boas test meal. Ehrenreich, in 1912, found that trypsin was present in the gastric contents of more than 50 per cent of 61 patients who were suffering from various gastro-intestinal disorders. Medes and Wright tested the contents of the stomach for 3 constituents of the duodenal fluid—bile, trypsin, and sucrose—and found evidence of duodenal regurgitation in all of 23 patients examined. Trypsin was most commonly found, often in the absence of bile, but bile was rarely present without trypsin. Spencer and his coworkers also found a tryptic enzyme to be present in the fasting or resting stomach in nearly all tests. The amount of bile, as estimated by the color of the fluid, varied considerably, but when the amount of bile was high the amount of trypsin was also increased.

Using the X-ray and an opaque meal, Bolton and Salmond examined 100 persons and actually saw regurgitation in 6. In the 94 remaining they saw antiperistalsis in the duodenum which they considered should be capable of causing regurgitation. Hicks and Visher in X-ray studies, noted rings of constriction and segmental contractions in the duodenum, and believed that these could force the duodenal contents back into the stomach if the pylorus were relaxed. These observers also introduced 150 cubic centimeters of 0.5 per cent hydrochloric acid into the stomach and found evidence of regurgitation in 38 per cent of 56 trials in dogs, and 40 per cent of 10 trials in man. They determined the presence of regurgitation only by the presence of bile as they did not consider that much duodenal content could be present in the stomach without it.

It is evident from a consideration of the above data obtained by various methods and by several different workers, that duodenal regurgitation is a factor which must be accepted. It is true that when one uses an acid meal instead of a non-acid meal of the Ewald type there is more regurgitation; in fact as the strength of the acid is increased there is, as

[1] From the Departments of Experimental Surgery and Physiology, Creighton University School of Medicine.

pointed out by Morse and by Medes and Wright, some increase in the amount of regurgitation. Indeed, this fact is one which gives the acid meal a unique value, since it gives one an index as to the capacity of the individual to regurgitate duodenal contents and lower excess acidity in the stomach.

With regard to the mechanism by which the reduction of acidity is accomplished, however, there is considerable difference of opinion. The mere fact that the acid has been reduced in strength is not proof that it has been neutralized, since another factor, that of dilution, must be taken into account. The preponderance of opinion at present is that neutralization brings about most of the reduction. Thus Olch attributes greatest importance to neutralization of the acid by regurgitated pancreatic juice. Elman insists that the amount of duodenal fluid necessary to dilute the acid meal is too great for the duodenum to secrete. He points out that to reduce the acid from 140 to 35 (total acidity), 3 volumes of neutral fluid would be necessary, whereas only ¾ volume of pancreatic juice would be required. He found that, using the "altercursive" method, little reduction occurred when the pancreatic juice was drained outside the body, but when the pancreatic juice was allowed to flow into the duodenum again, reduction became normal. Ortner, on the other hand, believes that dilution alone could account for the reduction noted.

In order to investigate the relative importance of dilution and neutralization we utilized a method of determining the amount of dilution which was first described by Gorham, and which we have already used extensively in studies on gastric acidity. The method consists, essentially, in adding to the acid test meal a coloring matter, so that when dilution occurs the color will decrease in intensity in direct proportion to the dilution. For this purpose phenolsulphonphthalein serves admirably since it is not absorbed by the stomach, and after it has been alkalinized, changes in its color can easily be read on a colorimeter. Details of the method and procedure used are as follows:

The experiments were performed in all cases on healthy dogs about 24 hours after the last feeding. A stock solution was prepared which consisted of 1,100 cubic centimeters of approximately tenth normal hydrochloric acid in which 12 milligrams of the sodium salt of phenolsulphonphthalein had been previously dissolved. The stomach was first lavaged with some of this solution in order to remove any fasting contents or mucus and then the test meal, which consisted of 300 cubic centimeters of the acid-phenolsulphonphthalein stock solution, was introduced. Samples were withdrawn from the stomach every 15 minutes for one hour, or as long as any fluid could be obtained. A portion of each sample and a portion of the original test solution were centrifuged, alkalinized by the addition of 1 cubic centimeter of a saturated solution of sodium hydroxide for each 10 cubic centimeters of the sample, and then centrifuged again. The phenolsulphonphthalein concentration of each sample was then determined by means of a colorimeter, using the alkalinized portion of the original test solution as a standard. In the occasional instances in which mucus interfered with the reading by causing turbidity the mucus was precipitated out by the addition of sodium tungstate and sulphuric acid.

The strength of the acid in the acid meal before its introduction into the stomach, and in the fractions removed from the stomach was determined by chemical analysis, since it was found that this method gave a more uniformly accurate result than titration. The acid chloride content of the test solution was determined directly, but in the samples removed from the stomach it was necessary to determine the total and neutral chloride, the acid chloride being the difference between these two figures.

The phenolsulphonphthalein reading indicates the percentage concentration of the original acid test solution which is present in the sample removed from the stomach. Consequently, the phenolsulphonphthalein reading multiplied by the chloride concentration of the original acid solution gives the acid chloride concentration which would be present in the sample at that dilution, if no neutralization has occurred. The acid chloride content of the sample has actually been determined,

Dog number	Chloride of acid meal	Hours after giving meal	Total chloride	Mineral chloride	Total minus mineral	P. S. P. per cent	Chloride of acid corrected for dilution	Reduction due to dilution	Reduction due to neutralization	Per cent reduction due to dilution	Per cent reduction due to neutralization
1	348	¼	345	15	330	99	344.5	3.5	14.5	19	81
		½	354	78	276	94	327	21	51	28	72
		¾	347	100	247	87	303	45	56	44	56
		1	349	129	220	83	289	59	09	46	54
2	348	¼	334	45	289	86	299	49	10	83	17
		½	332	85	247	77	268	80	21	79	21
		¾	332	131	201	72	251	97	50	66	34
3	348	¼	338	35	303	89	310	38	7	89	11
		½	339	59	280	87	303	45	23	66	34
		¾	343	91	252	83	289	59	37	61	39
		1	347	128	219	77	268	80	49	62	38
4	348	¼	342	21	321	93	324	24	3	90	10
		½	339	50	289	86	299	49	10	83	17
		¾	330	89	241	79	275	73	34	68	32
		1	325	134	191	05	226	122	35	78	22
5	353	¼	351	21	330	98	346	7	16	30	70
		½	355	40	315	92	325	28	10	71	29
		¾	354	55	299	91	321	32	22	59	41
		1	354	93	201	83	293	60	32	65	35
6	350	¼	348	30	318	93	326	24	8	75	25
		½	348	44	304	90	315	35	11	76	24
		¾	348	58	290	85	298	52	8	86	14
		1	351	103	248	81	284	66	36	65	35
								Average		65	35

The above chloride determinations are expressed in milligrams per 100 cubic centimeters.

however, and in all cases in this series it is seen to be lower than the calculated value of the acid meal corrected for dilution. The difference between this actual value and the value of the acid meal corrected for dilution therefore represents neutralization.

The total reduction in acidity (or in acid chloride concentration), which is represented by the difference between the acid chloride of the test meal and the acid chloride of the fraction removed from the stomach, can be divided therefore into two portions. The reduction due to dilution is the difference between the chloride of the test meal and the value which it would have at the dilution indicated by the phenolsulphonphthalein reading. The reduction due to neutralization is the difference between the chloride of the acid meal corrected for dilution and the actual acid chloride content of the sample.

The accompanying table shows the results obtained in a series of experiments, and these findings are typical of our results in a much larger number of animals.

SUMMARY AND CONCLUSION

The reduction in acidity which occurs when an acid meal is introduced into the stomach is due to both neutralization of the acid and dilution of the test meal by fluid which regurgitates into the stomach from the duodenum. We have outlined a method by which the amount of reduction due to dilution and the amount of reduction due to neutralization can be determined. It was found in a series of fractional tests made on 6 dogs that an average of 65 per cent of the reduction was due to dilution and 35 per cent due to neutralization.

REFERENCES

1. Bolton and Salmond. Lancet, 1927, 1:1230.
2. Ehrenreich. Ztschr. f. klin. Med., 1912, 75:231.
3. Ehrmann and Lederer. Deutsch. med. Wchnschr., 1909, 35:879.
4. Elman, R. Arch. Surg., 1928, 16:1256.
5. Gorham, F. D. J. Am. M. Ass., 1923, 81:1738.
6. Hicks and Visher. Am. J. Physiol., 1915, 39:1.
7. Medes and Wright. J. Clin. Investigation, 1928, 6:403.
8. Morse, W. E. Am. J. Physiol., 1916, 41:439.
9. Olch, I. Y. Arch. Surg., 1928, 16:125.
10. Ortner, A. Arch. f. d. ges. Physiol., 1917, 186:124.
11. Spencer, Meyer, Rehfus, and Hawk. Am. J. Physiol., 1915, 39:459.
12. Wilhelmj, Neigus, and Hill. Am. J. Physiol., 1933, 106:381.

CLINICAL SURGERY

FROM THE LAHEY CLINIC

THYROIDITIS

An Operative Procedure for the Relief of Tracheal Constriction Due to Thyroiditis

FRANK H. LAHEY, M.D., F.A.C.S., Boston, Massachusetts

SUBACUTE thyroiditis, even when thyroid tissue is almost completely replaced by round cell infiltration and inflammatory reaction, Riedel's struma, or when the thyroid gland is completely involved in the Hashimoto type of reaction called strumitis lymphomatosa, rarely causes sufficient tracheal obstruction so as to make an operative procedure necessary. Occasionally, however, patients with thyroiditis complain sufficiently of tracheal constriction as to justify an operation to give them relief.

In visualizing the cause of constrictive symptoms in chronic thyroiditis, one must recall that the thyroid gland surrounds at least two-thirds of the trachea (see Fig. 1 c) and that, as the result of the inflammatory process in the gland, its normally firm attachments to the trachea become even more firm, so that as the result of organization and cicatrization of the thyroiditis more and more constrictive pressure is made upon the trachea.

Not infrequently, X-ray pictures (Fig. 2) of the air-filled trachea will demonstrate in these cases of thyroiditis moderate but definite narrowing of the tracheal caliber at the point where the isthmus and body of the thyroid are in contact with the trachea. In such cases, should the patient complain of constrictive symptoms, it has been our custom when operation seemed necessary, to remove the isthmus of the thyroid so that the trachea was completely bared and the middle segment of thyroid entirely removed (Fig. 1 b).

I wish to add a step to this procedure which in no way complicates the operation but will overcome one of the serious complications which I

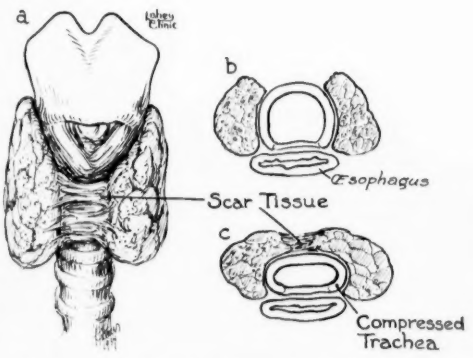


Fig. 1. a, This illustrates diagrammatically the isthmus excised and band of scar tissue joining the two cut ends of the thyroid stumps, thus additionally constricting the trachea. b, The break in the ring of thyroid tissue which exists after removal of the isthmus. c, The scar tissue union between the cut ends of the thyroid seen on cross section and indicating how such a cicatricial joining increases the constrictive effect of the thyroiditis.

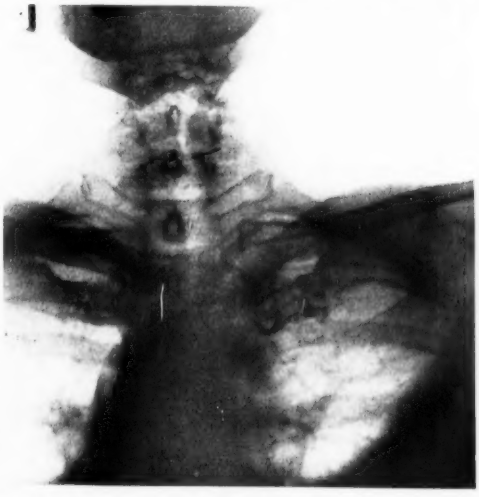


Fig. 2. An anteroposterior view of a trachea narrowed by the constrictive effect of thyroiditis.

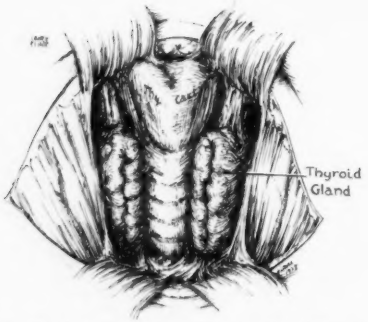


Fig. 3. The isthmus has been removed and the cut ends of the gland sutured. This is rarely possible owing to the woodlike firmness of the gland.

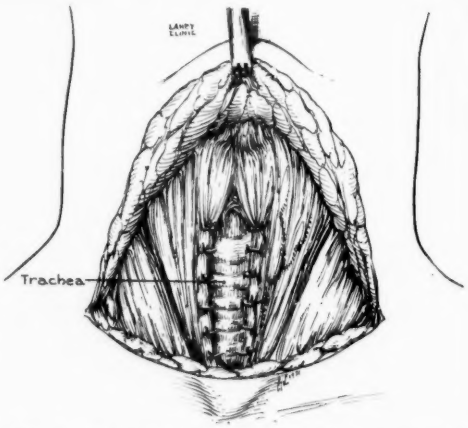


Fig. 4. The procedure herein suggested to prevent union of the two cut ends of the thyroid after removal of the isthmus. Note that all thyroid tissue has been removed from the trachea and that the sternohyoid muscles have been sutured to the trachea well out on its lateral walls so that there is a wide gap between their two bellies.

have seen occur with removal of the thyroid isthmus for constrictive symptoms.

The removal of the thyroid isthmus, as shown in Figure 1 b, breaks the connection between the two lobes of the thyroid gland and gives immediate relief from pressure symptoms. When, however, the prethyroid muscles are sutured together over the face of the gland, the two separated lobes tend either to pull together or to be joined by scar tissue (Fig. 1 a and c), thus re-establishing and intensifying the constrictive effect upon the trachea of the encircling, chronically inflamed thyroid gland. Due to the fact that a good sized segment of the constricting ring of organizing thyroid tissue has been removed and the ends allowed to re-establish their connection, this encircling ring of thyroid tissue has been markedly narrowed, and, because of this, effectually causes greater tracheal constriction (Fig. 1 c). As the result of such an operative procedure, we have seen the tracheal caliber further narrowed to the size of a millet seed so that the patient could not undertake any activity necessitating increased degree of air intake. I have employed this simple operative step (Fig. 4) to prevent possible reunion of the two cut ends of the thyroid isthmus.

If any patient with thyroiditis complains sufficiently of tracheal constriction and if a roentgenogram shows definite tracheal narrowing, we remove the entire thyroid isthmus well out to the points where it enters the lateral thyroid lobes. Care is taken to separate the thyroid isthmus completely from the trachea so that after its removal the trachea is left entirely free from any adherent thyroid tissue. All bleeding over the cut ends of thyroid tissue is controlled and, if possible, the cut ends are sutured over (Fig. 3). In most cases, owing to the rigidity of the infiltrated thyroid tissue, this cannot be satisfactorily accomplished. Whether or not it can be done is unimportant if suturing of the prethyroid muscles to the lateral walls of the trachea (Fig. 4) is employed.

Fine interrupted catgut stitches can be readily passed through the fascia over the trachea without entering the lumen of that structure, and the median edges of the sternohyoid muscles can be sutured to the trachea, thus covering the cut ends of the gland and interposing themselves between the cut ends of thyroid tissue so that the segments of thyroid tissue cannot reunite. Two points should be especially emphasized: (1) a wide segment of isthmus must be resected, and (2) the sternothyroid muscles must be attached to the trachea well down on its lateral walls so that a good sized gap is present between the two cut ends of the thyroid (Fig. 4). If one wishes to unite the two bellies of the prethyroid muscles so that the skin flap does not become adherent to the trachea and bob up and down on swallowing, stitches may be inserted further out in the bellies of the sternohyoid muscle and the muscles brought together after those structures have been so freed from their attachment to the sternomastoid borders that they will really displace toward the midline. This, however, is usually unnecessary. The procedure described, by interposing and fixing the muscle fibers between the two cut ends of thyroid tissue, establishes a gap, breaks the encircling constriction, and permanently insures against the re-establishment of the tracheal constriction.

THE CURABILITY OF MALIGNANT TUMORS OF THE UPPER JAW AND ANTRUM[1]

GORDON B. NEW, M.D., F.A.C.S., ROCHESTER, MINNESOTA
Section on Laryngology, Oral and Plastic Surgery, The Mayo Clinic

CLYDE M. CABOT, M.D., ROCHESTER, MINNESOTA
Fellow in Otolaryngology and Rhinology, The Mayo Foundation

IN the last two decades there has been marked improvement both in the treatment of malignant tumors of the upper jaw and antrum, and in the end-results. Twenty years ago, resection of the upper jaw was performed in such cases; some patients died following operation, and a small number of patients remained well 5 or more years after operation. In 1920, one of us (New) reported a method of treatment of malignant tumors of the upper jaw and antrum by means of cautery and irradiation, bringing out the fact that by this method of treatment operative mortality had been eliminated and the number of recurrences had been greatly reduced. Six years later, a second report was made of the end-results from treatment of 97 patients with malignant tumors of the upper jaw and antrum by means of surgical diathermy, cautery, and radium, and the fact was again emphasized that the end-results revealed that more patients remained well by this method of treatment than by methods previously used.

REVIEW OF LITERATURE

The literature on this subject recently has been very carefully reviewed in a monograph by Öhngren. He traced the development of electrosurgery and irradiation in the treatment of the condition on the Holmgren service of the Sabbatsberg Clinic at Stockholm. At first, the "soldering iron," or "ferrum candens," was used; large electric cauteries were then developed and, finally, electrosurgery. Öhngren reviewed both the foreign and American literature, and his monograph is an outstanding contribution to the subject. He has evolved a clinical classification of his own for determining malignancy of the tumor, in which he combines the position of the growth, with regard to whether indications for operation are favorable or unfavorable, its activity, determined microscopically, and the presence or absence of metastasis. He divides the skull, by means of an imaginary plane passing through the inner canthi and angles of the mandible, into an anterior-inferior or less dangerous portion and into a posterior-superior or more dangerous portion. In turn, each of these portions is further divided into four regions, two lateral and two medial. Tumors in the medial posterior-superior regions are much more dangerous and treatment is more likely to result in a smaller percentage of cures than with those in the lateral anterior-inferior regions.

In England, the method of treating these tumors also has undergone evolution, as is indicated by two symposiums in the Section of Laryngology of the Royal Society of Medicine in 1922 and in 1931. In the first discussion the consensus was that these tumors should be removed by surgical cutting methods, the operative technique was stressed, and the method of using cautery and diathermy was thought little of by practically all the members. In November, 1931, however, the discussion revealed that the values of diathermy and of irradiation were appreciated, and that resection by means of knife and scissors was little used.

A similar change in the treatment of these tumors has taken place in this country, as is indicated in articles by Sharp, by Quick, and by Houser. Most radiologists, however, make an opening into the antrum for purposes of drainage only, and they do not attempt to destroy the tumor, when that is advisable, with surgical diathermy.

DIAGNOSIS

A diagnosis of epithelioma of the upper jaw, when the growth originates in the mucous membrane, is, of course, not a difficult one. At times the condition is associated with papillary leucoplakia, a condition which potentially is malignant, and should be treated as such. Adenocarcinoma of mixed tumor type usually occurs posteriorly in the upper jaw and palate and, as a rule, presents a nonulcerated, smooth, hard surface. Fresh, frozen sections should be used in all cases to make a diagnosis at the time of operation, in order to determine the type of malignancy and its activity.

Patients with a constant complaint of some pain or uneasiness in the upper jaw or in the face, whose symptoms have been of short duration and who state that the pain or uneasiness has been made worse by lying down, should be carefully examined to exclude malignant tumor as the cause

[1] Read before the Canadian Medical Association, Calgary, Alberta, Canada, June 21, 1934.

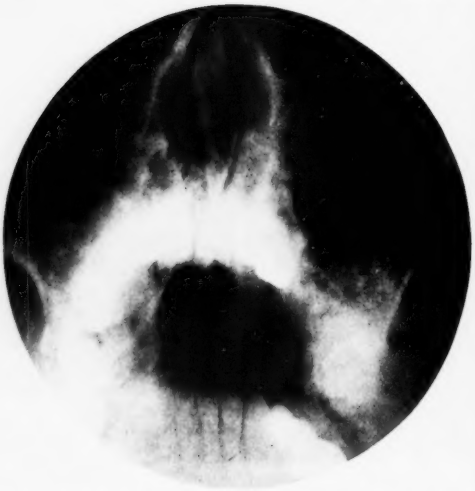


Fig. 1. Osteitis deformans of the upper jaw. The right antrum is involved.

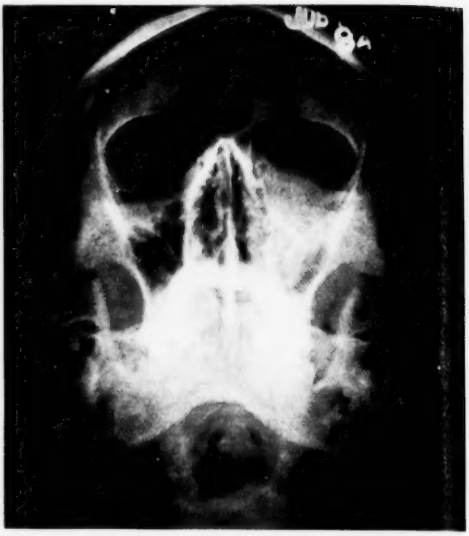


Fig. 2. Giant cell tumor of the right antrum.

of the pain. Roentgenograms should be made and careful study carried out to exclude any involvement of bone. In the indeterminate cases, with symptoms indicating a possible tumor of the antrum, it is advisable to explore the antrum above the alveolar process in order to make sure that the patient's pain is not due to a malignant condition in the antrum. This exploration should be carried out by the surgeon who is equipped to take care of the condition if it is found to be malignant, thus avoiding the necessity for two separate operations, first, exploration and biopsy and then, later, a second operation in which the malignant condition is cared for. Trauma associated with such a preliminary exploratory operation usually adds to the difficulties in determining the extent of the tumor at the second operation, and it is in these cases that recurrence is most likely to take place (Figs. 1 to 6).

Fig. 3. Fibromyxoma of the right antrum and upper jaw.

In the differential diagnosis of malignant tumor of the antrum, one must consider giant cell tumor, osteitis deformans, fibroma, adamantinoma, benign cyst, and osteoma. The roentgenogram is a great aid in determining, before operation, the type of tumor and its extent.

CASES STUDIED

We are presenting a study of 295 patients with malignant tumor of the upper jaw and antrum, who were operated on prior to January 1, 1929, in order to determine the curability of these tumors. There were 141 tumors of the antrum, of which 91 apparently were primary and 50 apparently secondary, and 154 tumors of the upper jaw. The division between primary and secondary tumors of the antrum is not absolute, since difficulty arose at times in determining whether the tumor originated in the jaw or in the antrum; the division is important, however, because of the difference in prognosis in the two groups.

Fig. 4. Fibro-osteoma of the left antrum and upper jaw.

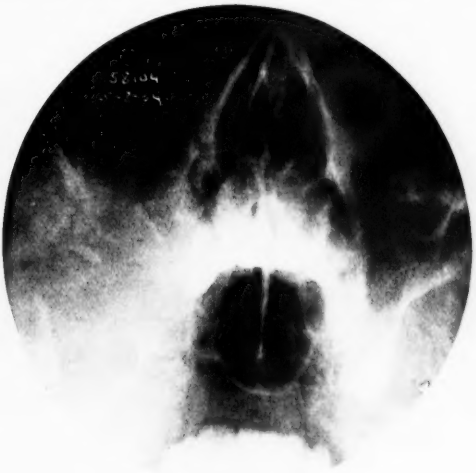


Fig. 5. Fibrosarcoma of the left antrum.

Of the 91 primary malignant tumors of the antrum, 63 were squamous cell epitheliomata; 6, adenocarcinomata; 7, round cell sarcomata; 9, fibrosarcomata or osteosarcomata; 3, lymphosarcomata; 2, malignant tumors of undetermined cell-type, and one was a myxosarcoma. The tumor was situated on the right side in 42 cases and on the left side in 49 cases, 46.2 and 53.8 per cent, respectively.

Of the 50 secondary malignant tumors of the antrum, 30 were squamous cell epitheliomata; 5, adenocarcinomata; 11, malignant tumors of undetermined cell type; 2, fibrosarcomata; 1 was a round cell sarcoma, and 1 a myxosarcoma. The tumor was situated on the right side in 25 cases and on the left side in 25 cases.

Distribution by age and sex. Of the 91 patients in the group with primary malignant tumor of the antrum, 48 were men and 43 were women, an approximately equal distribution between the sexes. The average age of the men in this group was 46 years and of the women 49 years. Of the 50 patients with secondary malignant tumor of the antrum, 44 were men and only 6, women, or 88 per cent compared to 12 per cent. The average age of the men in this group was 53 years and of the women 39 years. Of these 141 patients with primary or secondary malignant tumor of the antrum, 92 were men and 49, women, 65.2 per cent compared to 34.8 per cent. The average age of the men in both these groups was 49 years and of the women 47 years.

Malignant tumors of the upper jaw occurred approximately three times as often among the men in this group as among the women, 74 per cent, compared to 26 per cent. Of the 154 patients in the group, the average age of the men was 54 years and of the women 44 years.

Of the 295 patients in the series, 206 were men and 89 women, 69.8 per cent compared to 30.2 per cent. The average age of men in the series was 52 years and of women, 46 years.

Treatment. The selection of cases for operation is based on the situation and extent of the growth,

Fig. 6. Epithelioma of the left antrum.

TABLE I.—SURVIVAL AFTER OPERATION FOR VARIOUS CONDITIONS

Condition		Underwent operation			Lived after operation		
		Prior to	Patients	Traced	Patients	Per cent of traced patients	Years
Antrum	Primary malignant tumors	1-1-29	91	75	30	40.0	5 or more
		1-1-24	58	49	12	24.5	10 or more
		1-1-19	21	17	4	23.5	15 or more
	Secondary malignant tumors	1-1-29	50	43	23	53.4	5 or more
		1-1-24	47	35	12	34.3	10 or more
		1-1-19	16	12	3	25.0	15 or more
	Primary and secondary malignant tumors (total of the two foregoing divisions)	1-1-29	141	118	53	44.9	5 or more
		1-1-24	100	84	24	28.6	10 or more
		1-1-19	37	29	7	24.1	15 or more
Malignant tumors of upper jaw	Including involvement of palate	1-1-29	154	118	74	62.7	5 or more
		1-1-24	98	66	28	42.4	10 or more
		1-1-19	66	40	13	32.5	15 or more
	Without involvement of lymph nodes	1-1-29	136	104	71	68.3	5 or more
		1-1-24	84	55	27	49.1	10 or more
		1-1-19	56	32	13	40.6	15 or more
	With involvement of lymph nodes	1-1-29	18	15	4	26.7	5 or more
		1-1-24	14	12	2	16.7	10 or more
		1-1-19	10	9	1	11.1	15 or more
Primary malignant tumors of upper jaw and secondary malignant tumors of the antrum		1-1-29	204	161	97	60.2	5 or more
		1-1-24	140	101	40	39.6	10 or more
		1-1-19	82	52	16	30.8	15 or more
Primary and secondary malignant tumors of upper jaw and antrum		1-1-29	295	236	127	52.8	5 or more
		1-1-24	198	150	52	34.7	10 or more
		1-1-19	103	69	20	29.0	15 or more

the type of tumor, and the presence or absence of metastasis, and, in addition, the patient's age, general condition, and ability to return for observation. These patients should be followed up carefully after operation, so that if the growth recurs they can be treated immediately. Patients with extensive, primary antral tumors of highly malignant type have a better chance of recovery than those with tumors of the same size but of a low grade of malignancy, so that patients with very extensive, squamous cell epitheliomata, graded 4, or with sarcomata, are treated.

The treatment of tumors of this type has been outlined by us in previous reports. In most of our cases anæsthesia is by intratracheal administration of nitrous oxide, the tumor as a rule being approached through the mouth above the alveolar process or through the palate depending on its situation. Biopsy is made and a fresh frozen section is examined microscopically to determine the type of growth. In addition, lateral rhinotomy may be employed; an incision is made lateral to, and below, the nose, a portion of the nasal bone removed, and the growth destroyed through this approach. In the treatment of lesions of low grade, the aim is to destroy the tumor completely with diathermy, and with the more highly malignant tumors to establish thorough drainage by means of destruction by diathermy; radium is then inserted if it is required. A protected endotherm point, with the spark gap almost closed, is used in approaching the more dangerous regions about the orbit or the ethmoid bone, whereas a large point, with the spark gap well open, is employed in regions wherein wide destruction will not be of any serious consequence. Preliminary ligation of the external carotid artery is not done as a routine measure but is employed only in an occasional case in which secondary bleeding necessitates its use.

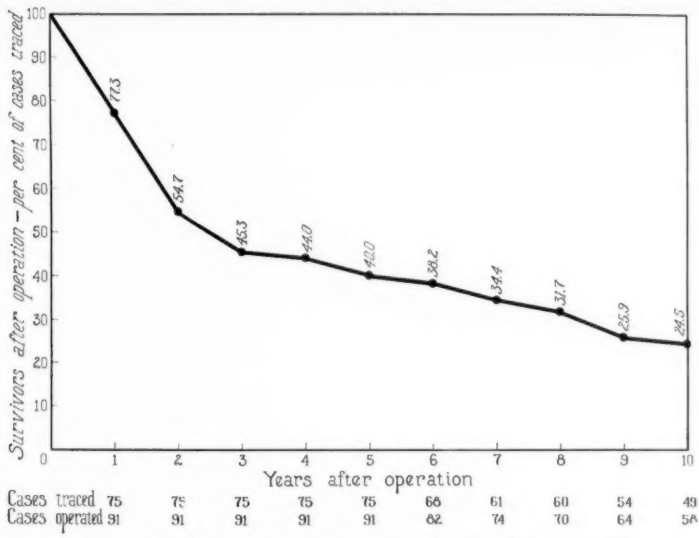


Fig. 7. Survival after operation for primary antral malignancy.

Postoperative observation every month or 6 weeks is advisable in cases of malignant tumor of the antrum. Sequestra are removed as soon as they tend to loosen. A vulcanite dental plate is used to close the postoperative opening in the upper jaw and is much preferable to a pedicled flap brought in from the neck. It is difficult to get a dental plate with artificial teeth that fits satisfactorily over the flap used to close a large postoperative opening in the jaw. Postoperative perforations in the cheek or nose, which occur in a small group of patients, are best closed by tubed flaps brought up from the neck and thorax. In most cases it is necessary to line the flap with a full thickness skin graft in order to obtain a double layer of epithelium and to get a flat graft. We prefer to raise a flap with the upper end in the supraclavicular region and the lower end extending down over the thorax. After delaying it the distal end is carried to the mastoid region, and, at the next stage, the proximal end, in the region of the clavicle, which has been lined with a full thickness skin graft, is brought to the area on the face where it is required. This method eliminates the scarring of the neck that follows the use of a tubed flap in the cervical region, as usually is employed. The character of the skin in the supraclavicular region is similar to that on the face and makes for a very satisfactory cosmetic result.

Results. The results of treatment of malignant tumors of the upper jaw and of the antrum are given in Table I. There was but 1 death in this series during the postoperative period of convalescence; this patient died of bronchopneumonia during an epidemic of influenza.

The results of Holmgren and Öhngren reveal 38.5 per cent (± 6.7) five year cures in 52 cases of malignant tumor of the antrum. They were able to trace all of their patients, whereas of 141 patients with primary or secondary malignant tumor of the antrum who were operated on prior to January 1, 1929, we were able to trace 118; of these, 53 were well without recurrence, which makes 44.9 per cent 5 year cures among patients who were traced, or 37.5 per cent of the number operated on. Houser reported 15 cases, with 13.3 per cent 5 year cures, and Peyton reported 11 cases; 2 of Peyton's patients are still living, 1 for more than 5 years (Figs. 7 to 10).

Of patients with primary malignant tumor of the antrum, 40 per cent of those who were traced were alive without recurrence 5 years after operation. Among patients with primary sarcoma of the antrum, 72.2 per cent of those who were traced were alive without recurrence 5 years after operation. The fact that many of the growths were of low-grade malignancy accounts for these results. Of patients with primary epithelioma of the antrum who were traced and the tumor graded, a higher percentage of those with tumors of low grade (grades 1 and 2) were alive without recurrence 5 years after operation than were those with tumors of high grade (grades 3 and 4), 55.6

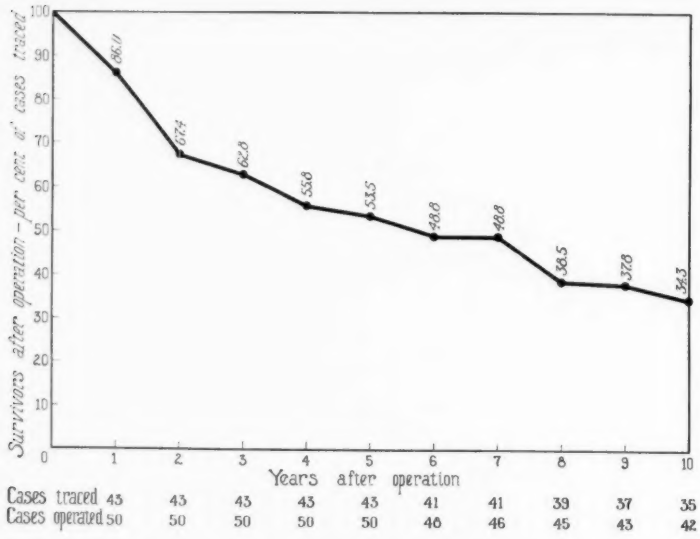


Fig. 8. Survival after operation for secondary antral malignancy.

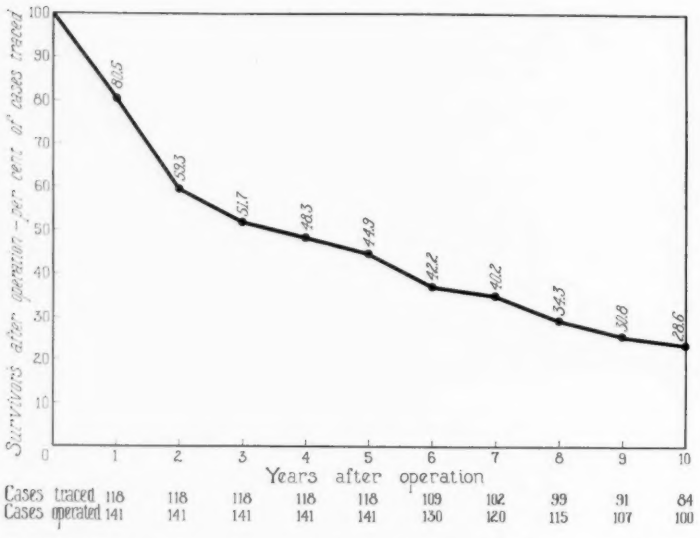


Fig. 9. Survival after operation; primary and secondary malignancy of the antrum, taken together.

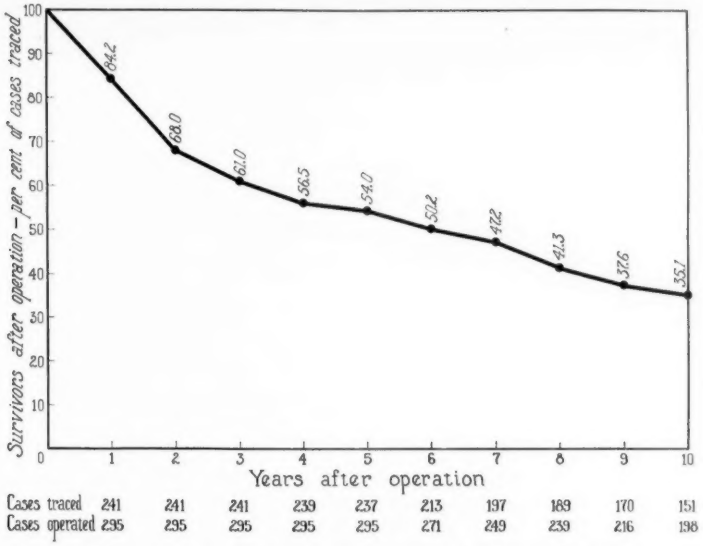


Fig. 10. Survival after operation for malignancy of the antrum and upper jaw.

per cent as compared to 34.5 per cent. Of patients with primary adenocarcinoma of the antrum, 33.3 per cent of those traced were alive and without recurrence 5 years after operation; although these tumors are of low grade of malignancy, they are not radiosensitive, and complete destruction with diathermy offers the best chance of treatment. Of patients with secondary malignant tumors of the antrum, 53.4 per cent of those traced were alive without recurrence 5 years after operation. These tumors originate in the upper jaw, and the prognosis is better than with primary malignant tumors of the antrum. Of patients with adenocarcinoma originating in the upper jaw and involving the antrum secondarily, 60.2 per cent of those traced were alive and without recurrence 5 years after operation. Of the entire group of patients with primary or secondary malignant tumor of the antrum, 44.9 per cent of those traced were alive and free from recurrence 5 years or more after operation.

Of patients with malignant tumor of the upper jaw including the palate, 62.7 per cent of those traced were alive and free of recurrence 5 years or more after operation. Of patients with malignant tumor of the upper jaw without involvement of lymph nodes, 68.3 per cent of those traced were alive and free from recurrence 5 years or more after operation, whereas of those with involvement of lymph nodes only 26.7 per cent of those traced were alive without recurrence 5 years or more after operation.

Of the entire group of 295 patients with malignant tumor of the upper jaw and antrum, 236 were traced, and of these, 127 patients, or 52.8 per cent, were alive without recurrence 5 years or more after operation.

CONCLUSIONS

1. Malignant tumors of the upper jaw and of the antrum are curable.

2. Surgical diathermy and irradiation are the most efficient measures of treatment.

3. Biopsy should be made in all cases at the time of operation as an aid in determining treatment.

CONGENITAL ARTERIOVENOUS FISTULÆ OF THE EXTREMITIES VISUALIZED BY ARTERIOGRAPHY

BAYARD T. HORTON, M.D., F.A.C.P.,[1] AND RALPH K. GHORMLEY, M.D., F.A.C.S.,[2] ROCHESTER, MINNESOTA

ARTERIOGRAPHY by means of thorotrast is a satisfactory way of "visualizing" arteriovenous fistulæ in the extremities, and it permits study of the situation and structure of such lesions before surgical exploration. The importance of the method is illustrated by the following reports, and, so far as we are aware, these represent the first cases of congenital arteriovenous fistula to be studied in this manner; in both cases surgical treatment was successful.

The surgical treatment of congenital arteriovenous fistula of an extremity hitherto has been extremely unsatisfactory, if one can judge from reports in the literature. This is due to the fact that, in the congenital type, abnormal communication between the arteries and veins is usually multiple, and for that reason extremely difficult to find at surgical exploration. According to Lewis, only 30 cases of congenital arteriovenous fistula of an extremity were recorded in the literature in 1930. In 19 of these cases, surgical exploration had been carried out in an attempt to close the fistula. This was followed by amputation of the involved extremity in 11 cases; in other words 58 per cent of the extremities found to be involved at exploratory operation were subsequently amputated (this percentage probably would be higher if follow-up information had been available in all of the cases). Surgical cure was obtained in only one case, and that was the case reported by Lewis of a woman who had an arteriovenous fistula in the region of the right elbow. It is possible that this represented an acquired rather than a congenital arteriovenous fistula, as there was a history of an injury which was followed 2 hours later by pain and by swelling in the right elbow. Then too, in this case, apparently only a single abnormal arteriovenous communication was found at the time of surgical exploration.

One of us (Horton) reported 23 cases of congenital arteriovenous fistula of the extremities which were observed at the Mayo Clinic from June 28, 1929, to May 11, 1931. Surgical explorations were not attempted in this series of cases. However, one of these patients, a man aged 44 years with a congenital arteriovenous fistula of the left leg, was successfully treated following 7 injections of quinine ethyl carbamate. This patient has remained well. In addition to the case reported by Lewis, Rienhoff, in 1924, and Reid, in 1925, reported cases of congenital arteriovenous fistula, involving the vessels of the left and right sides of the neck, respectively, in which the patients were successfully treated surgically.

Every subject with congenital arteriovenous fistula of an extremity should be studied by means of arteriography before surgical exploration is carried out.

Bare reports of the 2 following cases have been given by us before the staff of the Mayo Clinic.

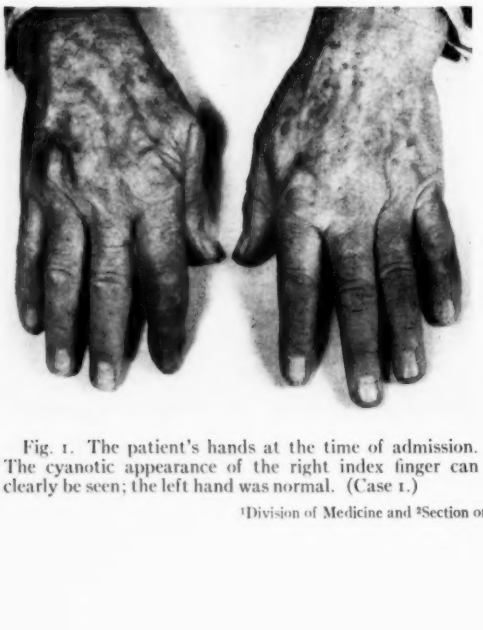


Fig. 1. The patient's hands at the time of admission. The cyanotic appearance of the right index finger can clearly be seen; the left hand was normal. (Case 1.)

REPORT OF CASES

CASE 1. A man, aged 60 years, came to the clinic December 4, 1933, because of pain and swelling of the right index finger, of about 2 years' duration. There was no definite history of injury to the finger which could account for his symptoms. April 9, 1933, he had sustained a fracture of the distal end of the right radius, but this had healed in a satisfactory manner without limitation of motion of the wrist joint.

The only significant finding on general physical examination was the condition of the right hand (Fig. 1). The patient was of normal build and weighed 195 pounds (88.5 kilograms). The blood pressure in millimeters of mercury was 170 systolic and 108 diastolic in the right arm, and 170 systolic and 110 diastolic in the left arm. The pulse rate was 70 beats per minute. There was spongy swelling of the index finger with beginning gangrene at the tip. The veins of this finger were definitely enlarged as compared with those of other digits. The maximal surface temperature of the right hand was higher by from 2.5 to

[1]Division of Medicine and [2]Section on Orthopedic Surgery, The Mayo Clinic.

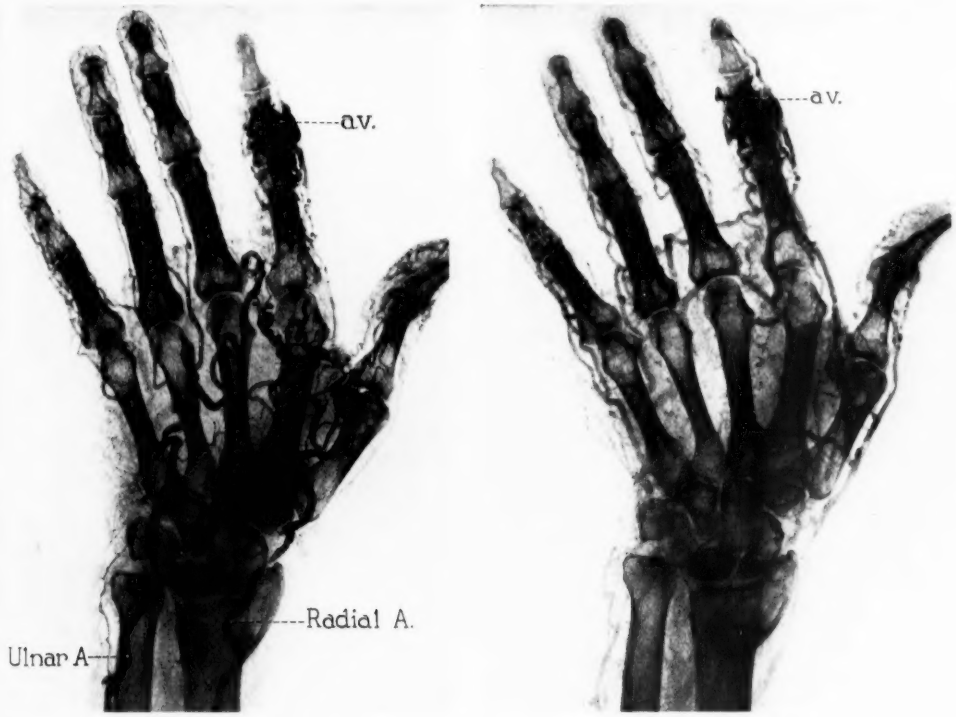


Fig. 2. Arteriogram of the right hand (December 6, 1933) after injection of an opaque substance into the brachial artery; a, the arterial tree is well outlined, but the opaque medium has not entered the distal portion of the right index finger; b, the venous tree is well outlined but that fistulæ in the index finger are present is evident. *av*, Showing the region in which the arteriovenous fistula was located. (Case 1.)

3 degrees C. than that of the left hand. Bradycardiac reaction could not be elicited when the right brachial artery was compressed by means of a sphygmomanometer cuff. A roentgenogram of the right hand revealed erosion of the lateral aspect of the middle phalanx of the index finger. A clinical diagnosis of arteriovenous fistula of the right index finger was made. This was confirmed by the finding of a high admixture of arterial blood in the veins of the right index finger and in those of the right hand. Blood removed under oil from a superficial vein of the right index finger, and from one of the right hand, disclosed an oxygen content of 20.25 and 20 per cent by volume, respectively, whereas blood removed in a similar manner from a vein of the left hand disclosed an oxygen content of 15.6 per cent by volume; the oxygen capacity was 21.15 per cent by volume. The oxygen content and capacity of the blood were determined by the Van Slyke gasometric method. The percentage of the saturation of oxygen in the blood was calculated by dividing the oxygen content by the oxygen capacity. The patient had been at rest in a horizontal position for 30 minutes when the specimens of blood were withdrawn. The oxygen saturation of blood from the right index finger was 95.8 per cent and that from the right hand 94.6 per cent. The oxygen saturation of blood from the left hand was 73.8 per cent. An arteriogram following injection of 10 cubic centimeters of thorotrast into the right brachial artery revealed evidence of an abnormal arteriovenous communication in the right index finger (Fig. 2, a and b).

The right index finger was amputated through the metacarpophalangeal joint, and the patient made a satisfactory recovery. The wound healed by primary intention. The

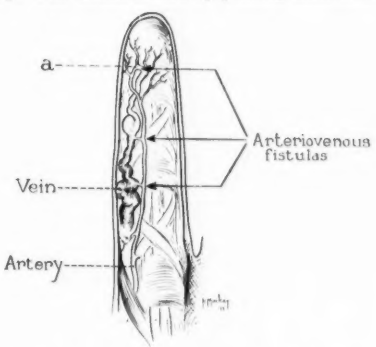


Fig. 3. A dissection of the right index finger illustrating three arteriovenous fistulæ. (Case 1.)

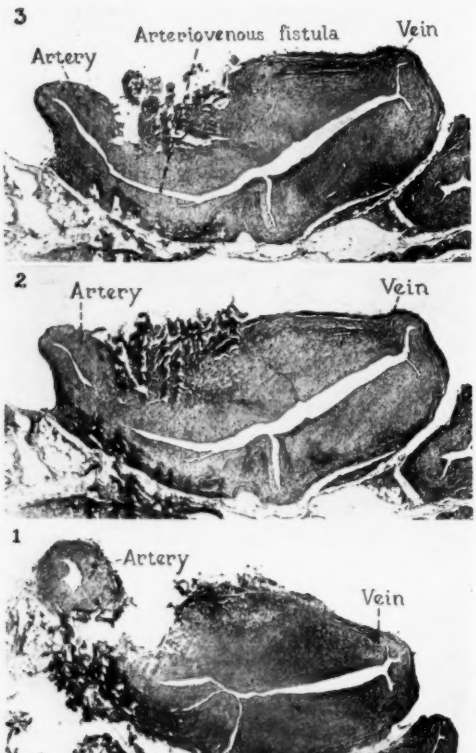


Fig. 4. Sections taken serially at point *a* in Figure 3; a direct communication between the artery and vein can be seen in the top section. (Case 1.)

vessels of the right finger were dissected, and at least 3 arteriovenous fistulæ were found between one of the digital arteries and an accompanying vein (Fig. 3). Microscopic sections (Fig. 4) taken serially at a point corresponding to *a* in Figure 3 revealed a direct communication between the artery and vein. The vein illustrated in this figure was large and tortuous, and there were many aneurysmal sacs along it. The walls of the veins in most of the microscopic sections were markedly thickened and these veins presented the general appearance of arteries. This was due to the fact that they were functioning as arteries.

Blood removed from veins of the right and left hands, 13 days after amputation of the right index finger, revealed the same concentration of oxygen in the venous blood of each hand. Forty-nine days after amputation of the right index finger, the systolic blood pressure in the right arm was 146 millimeters of mercury and the diastolic pressure, 90; in the left arm, the systolic pressure was 155 and the diastolic pressure 94.

We feel that the diagnosis of congenital arteriovenous fistula of the right index finger has been established in this case. This is the oldest patient with the condition who has been observed at the clinic. Lesions of this character, when confined to a hand or foot, almost invariably manifest themselves at birth, shortly after birth, or before the age of 20 years.

CASE 2. A man, aged 40 years in 1933, was observed at the clinic in December, 1926, February, 1928, December, 1929; and again in December, 1933. General examination has given essentially negative results at each of this patient's visits to the clinic, except for the condition in the right hand and forearm (Fig. 5). He is a strong, healthy looking individual, and routine laboratory studies have given negative results.

When he first came to the clinic a diagnosis of acquired arteriovenous fistula of the right hand was made, and the case originally was reported as an example of this condition by Yater. We are now of the opinion that this patient's condition was congenital, and one of us (Horton) included this case (as Case 22 in the table) in the report on congenital arteriovenous fistulæ published in 1932. The patient thought that his right hand had been injured catching a baseball when he was 20 years of age, and he attributed later developments to this injury. A break in the skin had not been observed at that time. The sudden development of a small arteriovenous fistula in the palm of the right hand at the age of 20 years could hardly account for the overgrowth of bone in the right forearm. The bones of the right forearm were 0.5 centimeters longer than the corresponding bones of the left forearm. Increase in length of a bone occurs apparently only when a fistula has been established for a considerable time before ossification of the epiphyseal cartilages takes place. It seems rather evident that this patient had had an abnormal arteriovenous communication in his right hand for a long time, and that his injury had merely aggravated the condition. The right middle finger had been amputated at the metacarpophalangeal joint in 1919, and the wound had healed promptly. On his first admission to the clinic in December, 1926, it was observed that the veins in the right forearm and back of the right hand were more prominent than those of the corresponding normal hand. There was a fluctuating, bluish mass, about 2.5 centimeters in diameter, at the base of the right fourth finger. This pulsated synchronously with the heart beat. No thrill was present, but a characteristic bruit was heard over the hand, and this bruit was accentuated with each heart beat. Compression of the ulnar artery caused the bruit to disappear; compression of the radial artery produced little effect on the bruit. This pulsating mass was explored by Dr. Pemberton in December, 1926, and the third and fourth digital branches of the superficial palmar arch and veins of the stump of the middle finger were ligated. About 8 weeks was required for the wound to heal. The same physical signs were present after the operation as before.

There had been very little change in the appearance of the hand at the time of the patient's second and third admissions, except that the pulsating region in the palm of the hand had increased slightly in size. This increase had been particularly noticeable during the 2 years preceding his last examination. At the last examination, the bruit was more marked than on previous visits, and there was a well developed, palpable thrill in the hand. This was evident when one shook hands with the patient, and the apex beat of the heart decreased from 3 to 5 beats per minute if the hand was gripped firmly. Compression of the ulnar artery did not cause the bruit to disappear.

Blood removed from a large vein of the right forearm

Fig. 5. Appearance of the hands and arms at the time of the patient's admission in December, 1933. Marked enlargement of the veins in the right hand and forearm may be noted. (Case 2.)

Fig. 6. Appearance of the right hand 16 days following amputation of the right fourth finger (amputation of the third finger had been performed at an earlier date). (Case 2.)

in 1928 (on the patient's second visit) had an oxygen saturation of 94 per cent. At the patient's last visit, blood removed from a vein at the right elbow had an oxygen content of 20.16 per cent by volume, and blood from a vein at the left elbow 12.49 per cent by volume. The oxygen capacity was 21.17 per cent by volume. The oxygen saturation of the blood in the right arm was 95.2 per cent, and from the left arm 59 per cent. The difference in the color of the blood from the two extremities was sufficient of itself to establish the diagnosis of an arteriovenous fistula, because the blood from the right arm was bright red, indicating its arterial character, whereas that from the left arm was dark red, such as one obtains from normal veins.

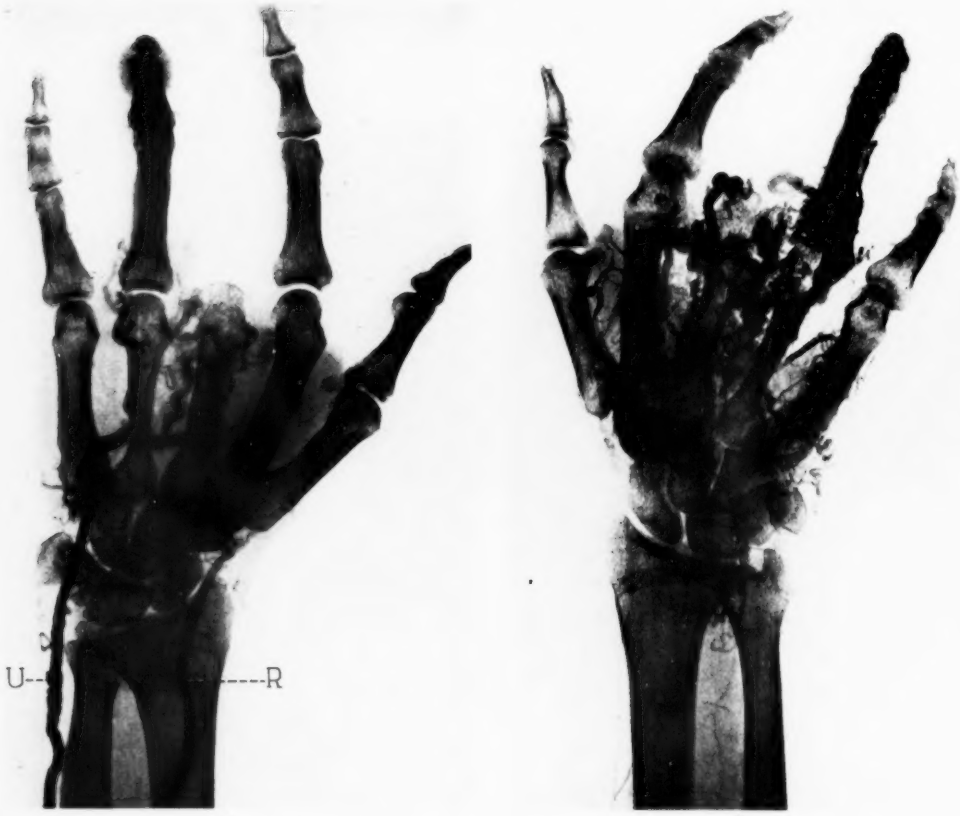
At the patient's first visit the roentgenogram of the right hand gave evidence of slight periarticular arthritis of the phalangeal joints, and this condition was still present at the last examination.

The maximal surface temperature of the right hand, at the last visit, was 4.7 degrees C. warmer than that of the left. The right hand liberated 101 small calories per minute, whereas the left hand liberated 92 small calories per minute. At the same visit, when the right brachial artery was compressed with a sphygmomanometer cuff, the apex beat of the heart decreased from 4 to 6 beats per minute. Blood pressure in the right arm in millimeters of mercury varied from 110 to 140 systolic and from 70 to 78 diastolic. On a previous visit (1928) a systolic blood pressure of 154 millimeters of mercury had been observed in the right arm. In the left arm, at the last visit, the systolic blood pressure varied from 105 to 134 and the diastolic blood pressure from 68 to 74.

An arteriogram of the right hand, made in 1933, following injection of 10 cubic centimeters of thorotrast into the right brachial artery revealed an interesting condition (Fig. 7, a and b). None of the opaque material which had been injected into the right brachial artery had entered the fingers; it had been short-circuited by way of the abnormal arteriovenous communications in the hand and had returned to the general circulation by way of the veins. One of us (Horton) in February, 1928, had observed the same phenomenon in this case following injection of 10 cubic centimeters of lipiodol into the right brachial artery.

December 21, 1933, the right fourth finger and a V-shaped portion of the palm of the right hand, including most of the third and fourth metacarpal bones, were removed surgically by one of us (Ghormley). The wound healed by primary intention in 16 days. The circulation in the thumb, index, and fifth fingers returned to normal, and the surface temperature of these digits remained essentially the same as that of the digits of the left hand (Fig. 6). Microscopic sections taken serially through the V-shaped portion of the palm of the right hand revealed many direct communications between the arterioles and venules.

The blood pressure on the first postoperative day was 142 millimeters of mercury systolic and 90 diastolic in the right arm, and 130 systolic and 65 diastolic, in the left arm. On the eighth postoperative day, the blood pressure in the right arm was 135 systolic and 75 diastolic, and in the left arm 100 systolic and 60 diastolic; at this time oxygen determinations of blood from a superficial vein at the right elbow revealed oxygen saturation of 89.9 per cent, and for blood from the corresponding vein of the left elbow oxygen saturation of 82.2 per cent. On the twentieth postoperative day, the mean blood pressure in the right arm was 115 systolic and 82 diastolic, and in the left arm 117 systolic and 84 diastolic. Blood removed from a superficial vein at the right elbow on the twenty-second postoperative day was of an oxygen saturation of 81.2 per cent and that from a corresponding vein of the left elbow 77.7 per cent. It is interesting to note that the oxygen saturation of blood from the right arm was slightly higher than that of blood from the left arm following surgical amputation of the right fourth finger and excision of a V-shaped portion of the palm of the right hand. This probably indicates that the patient still had additional, small, abnormal arteriovenous communications in the right upper extremity. Bradycardiac reactions could not be elicited during this latter period of observation.

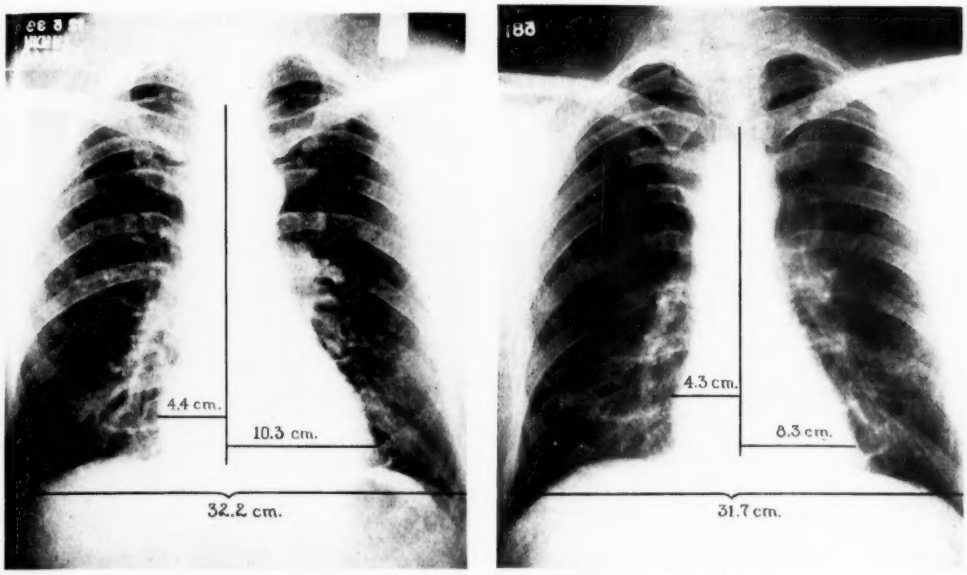


a. b.

Fig. 7. Arteriogram of the right hand (December 6, 1933) after injection into the brachial artery of an opaque substance; arteriovenous fistulæ may be seen. The opaque medium has not entered the digits, but has been short circuited by way of the fistulæ; a, arterial tree and b, venous tree; *U*, ulnar artery; *R*, radial artery. (Case 2.)

Since the patient's dismissal, he has written that he is able to use his right hand in plowing and routine farm work. Before the operation his hand was practically useless.

Some interesting clinical observations were made during the period of convalescence in Case 2. Teleoroentgenograms of the heart taken at the time of last admission revealed the transverse diameter to be 14.7 centimeters (Fig. 8a), but, 21 days following the operation, the transverse diameter of the heart had decreased to 12.6 centimeters (Fig. 8b), an actual decrease of 2.1 centimeters. It was evident that the arteriovenous fistulæ in the right hand had been of sufficient size to produce systemic effects on the cardiovascular system. The blood pressure had been slightly higher in the right arm than in the left, the heart rate had been consistently more than 80 beats per minute and when the right brachial artery was compressed by means of a sphygmomanometer cuff, it decreased from 4 to 6 beats per minute. It seems probable that the chief cause of the increase in size of the shadow of the heart was dilation with a minimal amount of hypertrophy, although one cannot be entirely sure of this. The decrease in size of the shadow of the heart was chiefly confined to the left ventricle, which is evident when one compares the teleoroentgenograms. This is what one would expect following surgical closure of arteriovenous fistulæ, such as were present in the right hand of this patient. It is interesting also to note that the total transverse diameter of the shadow of the heart in each instance was less than 50 per cent of the transverse diameter of the thorax.



a. b.

Fig. 8. a, Teleoroentgenogram of the thorax December 5, 1933, illustrating the size of the heart; b, January 11, 1934, showing definite decrease in the size of the heart. The measurements are of the cardiac shadow. (Case 2.)

Oxygen determinations of the venous blood serve not only to determine whether the patient has an arteriovenous fistula prior to surgical exploration, but also, when carried out following surgical operation, to give accurate information as to whether abnormal arteriovenous communications in the extremity have all been closed. This is a simple procedure and a valuable aid in estimating the results of operation. Without oxygen determinations of this character, one would have said that a perfect cure had been obtained in Case 2. This, however, is probably not true, although from all other clinical standpoints it would appear so.

SUMMARY AND CONCLUSIONS

Arteriography, such as was carried out in these 2 cases, offers an important means of finding abnormal communications between arteries and veins. One of us (Horton) previously has used this method to demonstrate an acquired arteriovenous fistula in the right thigh following a gunshot wound; this case has been recognized in the literature as the first in which an acquired arteriovenous fistula was visualized by means of arteriography. So far as we are aware, the 2 cases herein described are the first cases of congenital arteriovenous fistula in which the condition was visualized by arteriography. Visualization of arteriovenous fistulæ in the extremities, of both the congenital and the acquired types, can be carried out successfully in most instances and should be of great help to the surgeon in treating such lesions. At present it is only by means of arteriography that the surgeon can learn in which cases operation will be a remedial measure. Therefore, it is only by means of arteriography that additional progress will be made in the surgical handling of these vascular anomalies.

BIBLIOGRAPHY

1. Horton, B. T. Hemihypertrophy of extremities, etc. J. Am. M. Ass., 1932, 98: 373–377.
2. Idem. Arteriovenous fistula involving the common femoral artery identified by arteriography. Proc. Staff Meet. Mayo Clin., 1933, 8: 189–191.
3. Horton, B. T., and Ghormley, R. K. Congenital arteriovenous fistula. Proc. Staff Meet. Mayo Clin., 1933, 8: 773–776.
4. Lewis, D. W. Congenital arteriovenous fistulæ. Lancet, 1930, 2: 621–629; 680–686.
5. Reid, M. R. Studies on abnormal arteriovenous communications, acquired and congenital. I, Arch. Surg., 1925, 10: 601–638. II, Ibid., 996–1009. III, Ibid., 11: 25–42. IV, Ibid., 237–253.
6. Rienhoff, W. F. Congenital arteriovenous fistula, etc. Bull. Johns Hopkins Hosp., 1924, 35: 271–284.
7. Yater, W. M. Acquired arteriovenous fistula: report of four cases. Ann. Surg., 1928 87: 19–31.

RESECTION OF THE KIDNEY[1]

HERMAN L. KRETSCHMER, M.D., F.A.C.S., CHICAGO, ILLINOIS

IN view of the fact that the general tendency in the surgical treatment of various lesions of the kidney has had a distinct trend toward conservatism, and because resection of the kidney is an operation of great possibilities in well selected cases, it does seem strange that the operation has been performed relatively infrequently. In recent publications, both Young and Judd call attention to the fact that the operation is rarely performed.

With the development of modern urology and with the many problems calling for solution, particularly from the diagnostic standpoint, it is not surprising that this conservative procedure should temporarily be lost sight of.

Kuester, in his monograph published in 1896, collected but 30 cases that had been subjected to a resection of the kidney, with a mortality of 16.6 per cent. Perhaps resection fell into temporary disrepute because of the poor results obtained when the operation was first performed. Moreover, during the inceptive period of kidney resection, the operation was unwisely invoked, a matter clearly evidenced by the statistics of Kuester; in 16 of the 30 cases, resections were either for tumor or tuberculosis. The lack of refinements of diagnosis in some of the early cases must be held responsible for the failures recorded, since the operation was carried out in lesions which we now know are wholly unsuited for resection, among these lesions being malignant tumors and tuberculosis, conditions requiring a radical operation and not a conservative one.

Resections of portions of normally formed kidneys have been done for various pathological conditions and many of them were carried out long before the advent of modern urological diagnosis. Among the various lesions for which resections were done may be mentioned the following:

Spiegelberg (1870) accidentally resected an echinococcus cyst of the right kidney; Wells (1884) accidentally removed about one-third of the left kidney in shelling out a tumor in the region of the kidney; Czerny (1887) resected a tumor, an angiosarcoma of the kidney; Keetley (1890) removed, by lumbar incision, the loose and crushed-off lower extremity of the left kidney after a wagon accident; Waitz (1891) did a resection for pyonephrosis; Bardenheuer (1891) did resections in 2 cases—one for calculus pyonephrosis, the other for injury; Kummell (1893) reported 3 cases of resection—the first a localized inflammatory process (a hard nodule the size of a walnut), the second an abscess caused by a calculus, the third an echinococcus disease; Block (1896), resection of a tumor of the kidney, an adenoma; Moynihan (1902) reported 3 resections—(1) a solitary cyst; (2) a solitary cyst in a connecting band of a horseshoe kidney, (3) a tumor; Rathbun in a recent publication reported a series of six resections.

Resections of double kidneys have been performed more frequently than have resections of normally formed kidneys, but this is undoubtedly due to modern urological diagnosis. The lesions are generally infected hydronephroses with or without stone. In this connection it might be well to mention the case of Eisendrath, Phifer, and Culver, in which the lesion was bilateral and resection on each side was carried out.

In a discussion of resection of the kidney it is necessary to differentiate between two types: (1) resection of a part of a normally formed kidney; (2) resection of one-half of a double kidney.

In this series 10 resections were done upon 10 normally formed kidneys as distinguished from double kidneys. The resections were done for the following lesions:

	Cases
Stones	5
Solitary cyst	1
Solitary cyst and stone	1
Traumatic cyst	1
Benign tumor	1
Carbuncle	1

Resection of one-half of a double kidney was done in 6 cases. It has frequently been stated that kidneys which are the seat of congenital anomalies are more prone to undergo pathological changes than are normal kidneys. The 6 resections of double kidneys were done for the following lesions:

	Cases
Hydronephrosis	3
Hydronephrosis and stones	1
Hydronephrosis and ectopic ureter	1
Hydronephrosis and stone in ureter	1

INDICATIONS

1. Whether the solitary kidney is congenital in origin or the result of a previous nephrectomy, the problem of treating it when it becomes the seat of a serious lesion is the same. Among the lesions in a solitary kidney that are amenable to resection may be mentioned circumscribed collections of

[1] Read at the annual meeting of The American Association of Genito-Urinary Surgeons, Hot Springs, Virginia, May 14–17, 1934.

Fig. 1. Pyelogram after resection in Case 1.

Fig. 2. Pyelogram showing the presence of a large cavity in lower pole of kidney. Stones found in this cavity. Case 2.

stones, especially with infection, the presence of a stone in a calyx with dilatation and with a stricture of the *neck*, a hydronephrosis with more or less destruction of parenchyma, and encysted stones with abscess formation that leads to destruction of the parenchyma. Naturally, when these conditions are present in one of two kidneys, resection may just as well be performed.

Resection is also indicated—

2. In cases of bilateral stone in which one pole is diseased, or in cases in which there exists a circumscribed stone with pus formation;

3. In cases of partial hydronephrosis of the upper or lower pole with stone formation;

4. In cases of solitary cyst of the kidney. In this group of cases resection is at present recognized as the operation of choice and, as a rule, is very simple and easy of execution;

5. In cases of double kidney with double pelvis in which one-half shows the presence of hydronephrosis, severe infection, or stone;

6. In certain cases of metastic kidney infection, that is, carbuncle of the kidney. Resection in a case of this sort proved to be a very wise choice in which the opposite kidney became the seat of a carbuncle. This case (Case 10) had many interesting phases;

7. In case of poor function of opposite kidney.

In Table I is given a summary of the ages of the patients upon whom a resection of the kidney was done.

TABLE I.—AGES

Age in Years	Cases
Under 10	3
10 to 20	1
20 to 30	3
30 to 40	2
40 to 50	3
50 to 60	3
Over 60	1

Oldest patient, 61 years; youngest patient, 6 years.

GROUP I.—NORMALLY FORMED KIDNEYS, RESECTION

Recurrent Right Renal Calculi

CASE 1. D. W. R., male, aged 46 years, was admitted to the Presbyterian Hospital, July 8, 1928. The symptoms began 22 years ago, with left renal colic. The attacks would subside with the passage of gravel. Patient would then remain symptom free for a period of 4 to 5 years when another attack would occur. He also experienced attacks of colic on the right side. Twelve years later, after having received injections of oil in the left ureter, a stone passed. Two years later stones were removed from the right kidney.

For the past 5 years and up to the present admission to the hospital, the patient has complained of a pain in the right kidney region for which he sought relief.

Physical examination was negative. Urinalysis showed no sugar, no albumin, some pus. Examination of blood showed: red cells 4,850,000, leucocytes 9,900, hæmoglobin 88 per cent. Blood chemistry revealed urea nitrogen, 12.2; uric acid, 2.8; creatinin, 1.5, non-protein nitrogen, 37.2. The blood pressure was 122/72. The Wassermann reaction was negative. Roentgen-ray examination showed within the right kidney area a large, irregular, dense area, apparently a kidney stone filling the pelvis, and two shadows in the lower pole. Cystoscopic examination revealed a mild cystitis. The bladder was otherwise negative. Both ureters were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed the following:

	Leucocytes per cu. mm.	Cultures
Bladder	66	Bacillus proteus
Right kidney	21	Bacillus proteus
Left kidney	13	Sterile

Fig. 3. The peculiar shape of the stone in the lower pole of the kidney is shown. At operation a large cavity with a narrow neck was found. Case 3.

By thalein test the output was 74 per cent of the dye for 1 hour.

Operation was done July 11, 1928, under ethylene anæsthesia, and consisted of: (1) pyelotomy and removal of a large stone from the pelvis; (2) resection of the lower pole of the kidney. In the resected piece of kidney a very large cavity was found that contained two stones.

(The recurrent stones were probably due to the fact that when the stones were previously removed a cavity was left so that the patient again developed kidney stones.)

Result—uneventful recovery.

Subsequent X-ray examinations were negative for stone (Fig. 1).

Left Renal Calculi

Case 2. H. W., male, aged 30 years, was admitted to the Presbyterian Hospital, June 26, 1929. Attacks of pain in the left kidney region associated with nausea and vomiting began 7 years ago. The pain radiated along the course of the ureter. No hæmaturia, no frequency of urination, no nocturia were noted. X-ray examination at that time showed a stone in the left ureter which the patient passed 2 weeks later. The patient had no further trouble until 2 weeks ago when he was seized with a pain over the left kidney region which lasted for 24 hours.

Physical examination was negative. Urinalysis showed no sugar, no albumin, some blood, an occasional leucocyte and many red blood cells. Examination of blood showed: red cells 4,720,000; leucocytes, 12,000; hæmoglobin, 85 per cent. Blood chemistry examinations showed urea nitrogen, 18.3; uric acid, 3.9; creatinin, 1.9; non-protein nitrogen, 39.7. The Wassermann test gave a negative reaction. Roentgen-ray examination revealed a group of shadows in the region of the lower pole of the kidney on the left side. Cystoscopic examination disclosed a normal bladder. Both ureters were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed:

	Leucocytes per cu. mm.	Casts	Cultures	Tubercle bacillus
Bladder	50	0	Sterile	0
Right kidney	20	0	Sterile	0
Left kidney	80	Few granular	Sterile	0

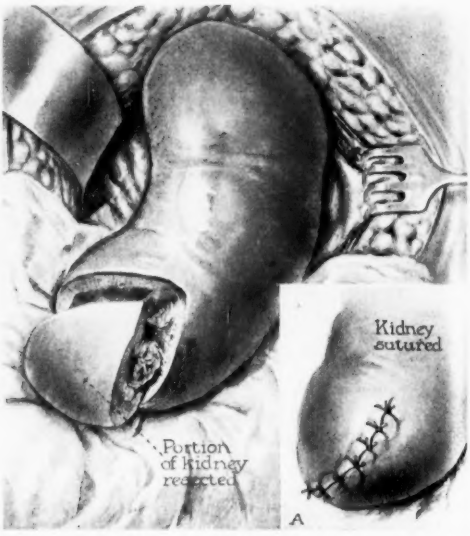


Fig. 4. A wedge shaped piece of kidney tissue containing a large cavity and a dumb-bell shaped stone was resected. Insert shows the line of suture. Case 3.

Guinea pig tests.—Bladder, right and left negative for tuberculosis. The left pyelogram (Fig. 2) showed a rather large pelvis; the calyces clubbed; the inferior calyx broad, not so densely filled as the rest, and with some mottling due to the stone shadows.

Operation was done July 8, 1929, under nitrous oxide and ether anæsthesia and consisted of resection of the lower pole of the kidney. This contained a large cavity in which 8 stones were found.

Result: uneventful recovery.

Subsequent roentgen-ray examination showed a complete absence of the nest of calculi previously shown near the lower pole of the kidney. The last (March 16, 1934) examination showed no stone shadows.

Blood chemistry examination, May 10, 1934 showed: urea nitrogen, 30; creatinin, 1.71; non-protein nitrogen 52.

Renal Calculi and Ureteral Calculi

Case 3. Dr. P. P., male, aged 34 years, admitted to the Presbyterian Hospital, November 18, 1927. He has had attacks of bilateral renal colic extending over a period of 15 years, during which time over 20 stones were passed. The attacks were associated with frequency, nocturia, urgency, burning, and intermittent hæmaturia. Seven years before he came under my observation, stones were removed from the right kidney. Two years later X-ray examination showed a recurrence of stones. The attacks of renal colic and the passage of stones have continued up to the present time.

Physical examination was negative.

Urinalysis showed no albumin, no sugar, no blood, leucocytes 50 per cubic millimeter.

Blood examination revealed red cells, 4,786,000; leucocytes, 14,400; hæmoglobin, 90 per cent. The blood pressure was systolic 118, diastolic 78. Roentgen-ray examination showed stones in both kidneys and two low-lying left ureteral stones.

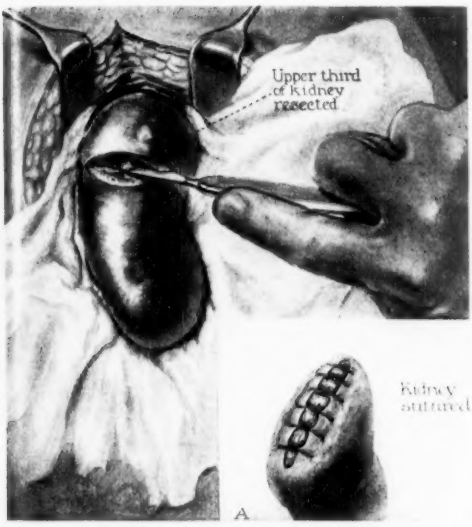


Fig. 5. Resection of the upper third of the kidney. The resected portion of the kidney contained a large cavity with multiple stones. Insert shows the line of closure. Case 4.

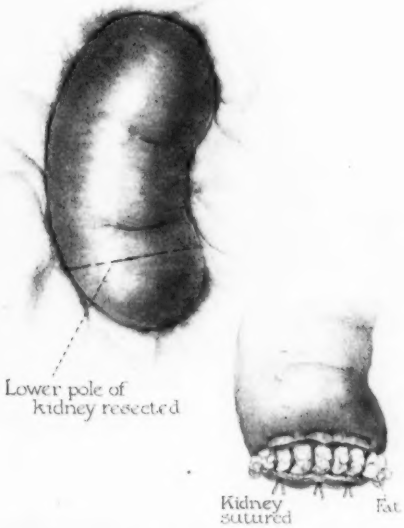


Fig. 6. Resection of lower pole of kidney. Insert shows the line of suture. Case 5.

Cystoscopic examination of the bladder was negative. The right and left ureters were catheterized without difficulty or obstruction. The urine obtained at this time showed:

	Leucocytes per cu. mm.	Cultures
Bladder	4	Sterile
Right kidney	3	Sterile
Left kidney	90	Sterile

Thalein test revealed a total output of 2 per cent from the right side and 5 per cent from the left side in 20 minutes. Roentgen-ray examination disclosed a large stone in the lower pole of the left kidney and 2 stones in the pelvic portion of the ureter. The stone in the kidney consisted of a very large lower part and an isthmus which was connected with a smaller part of the stone. This stone may be said to have a dumb-bell configuration (Fig. 3).

Over a period of 4 months oil injections were made through a ureteral catheter and during this time one of the stones was passed. The attacks of renal colic recurred more frequently and became more severe. Operation was advised.

On March 16, 1928, under ethylene anæsthesia a ureterotomy was done and the stone removed from the left ureter. A second operation, was done March 30, 1928, and ethylene anæsthesia was again used. At this time the lower pole of the left kidney was resected (Fig. 4).

(It seemed to me that resection of the kidney was clearly indicated because of the large cavity with a narrow neck that would have remained if only the stone had been removed.)

Result: uneventful convalescence.

Multiple Renal Calculi (Right)

Case 4. C. C., male, aged 12 years, was admitted to the Children's Memorial Hospital, November 9, 1932. At the age of 4 months the infant had entered the hospital because of difficulty and pain on urination for which a circumcision was done. When admitted to the hospital, he was suffering from an abdominal pain of the right side, nausea, vomiting, and fever. At this time a diagnosis of acute appendicitis was made.

Physical examination revealed a generalized rigidity of moderate degree over the entire abdomen, but especially over the upper right quadrant; tenderness over the entire right side, and a point of *severe* tenderness 1.5 centimeters above McBurney's point. Urinalysis revealed albumin +, acetone ++, benzidine ++, 10 to 12 red blood cells per high power field, an occasional leucocyte, numerous cystin crystals, no casts; no bacteria in stained specimen. Blood examination disclosed leucocytes, 19,650; polymorphonuclears, 84 per cent; lymphocytes, 11 per cent. Roentgen-ray examination showed two stone shadows opposite the transverse process of the first lumbar vertebra on the right side. An intravenous pyelogram revealed a slight delay in visualization of the dye on the right side and dilatation of the pelvis and ureter. The kidney and ureter on the left side were normal. There was, however, an increased density of the shadows in the region of the stones in the right kidney.

Operation was done November 25, 1932, and ether anæsthesia was used. The upper third of the kidney was resected (Fig. 5), and in it was found a large cavity, which contained stones and it had a narrow outlet. Chemical examination of the stones showed pure cystin. Subsequent examinations of the urine during patient's stay in the hospital showed no cystin.

Result: uneventful recovery. Patient was discharged, December 12, 1932.

Examination of the urine, April 27, 1933, again showed cystin crystals.

Chemical test for cystin was positive.

Fig. 7. Intravenous pyelogram showing deformity of the pelvis with mesial displacement of the ureter and outline of the cyst. Case 6.

Multiple Kidney Stones with Dilatation of the Inferior Calyx

Case 5. Dr. H. R., male, aged 39 years, was admitted to the Presbyterian Hospital, June 2, 1932. His first attack of right renal colic began 15 years ago. Subsequent attacks were severe and required morphine for relief. Following the second attack, a stone was passed. A roentgenogram 3 years ago showed three stones in the right kidney. During the past 3 years there has been a fairly constant right lumbar backache. Two or three mild attacks of chills with fever have occurred during the past 2 years and varying amounts of pus and an occasional red blood cell have been found in the urine.

Physical examination disclosed tenderness on deep pressure in the right costovertebral angle. The lower pole of the right kidney was palpable and tender. Bilateral incomplete direct inguinal hernia was noted. Urinalysis showed no albumin; no sugar; no blood; leucocytes, occasional; no red blood cells or casts were seen under the microscope. Examination of blood showed red cells, 5,120,000; leucocytes, 8,600; hæmoglobin, 90 per cent. The blood pressure was systolic 192, diastolic 90. Roentgen-ray examination disclosed 3 stones in the region of the lower pole of the kidney. Two of the stones were covered by the shadow of the last rib. The thalein test showed total output of dye, 56 per cent for 1 hour.

Operation was done June 3, 1932, and ethylene anæsthesia was used. Resection of the lower pole of the kidney revealed a large cavity containing stones (Fig. 6).

Result: uneventful recovery.

Subsequent roentgen-ray examination, January 10, 1933, revealed no signs of calculi at any point. The right kidney outline looked the same as in the picture of a year ago.

Blood chemistry examination, May 10, 1934, showed: urea nitrogen, 14; uric acid, 5.4; creatinin, 1.4; non-protein nitrogen, 33.

Fig. 8. Large solitary cyst springing from lower pole. Insert *A* shows the appearance of kidney after resection of cyst; insert *B* the line of closure. Case 6.

Solitary Cyst

Case 6. Mrs. E. G., aged 45 years, was admitted to the Presbyterian Hospital, June 1, 1932. In June, 1930, a vaginal hysterectomy was done, and the following October patient noticed in the left side of the abdomen at the level of the umbilicus a dull aching pain that radiated through to the back. The pain was intermittent; for weeks at a time it was completely absent, and then recurred and remained constant for several days or weeks. After an interval of 2 months, during which the pain was absent, a similar attack occurred, more severe in character. There were no urinary symptoms.

Physical examination was negative except for a cystic swelling of the thyroid and an enlarged left kidney which was smooth, not tender, and freely movable. The right kidney was not palpable. Urinalysis revealed no albumin; no blood; no sugar; but a few red blood cells present in the sediment. Blood examination disclosed red cells, 4,360,000; leucocytes, 11,100; hæmoglobin, 84 per cent. Blood chemistry showed urea nitrogen, 11; uric acid, 3.1; creatinin, 1.4; non-protein nitrogen, 34. The blood pressure was systolic 134, diastolic 76.

Roentgen-ray examination was negative for stone. A large swelling was visible which was attached to the lower pole of the left kidney. The findings were compatible with the finding of a solitary cyst which sprang from the lower pole of the kidney.

An intravenous left pyelogram showed the pelvis and calyces pushed up by the tumor mass in the lower pole so that the calyces were compressed in the medial portion and were drawn outward at the upper portion of the upper pole (Fig. 7)

Right Pyelogram.—Normal.

Cystoscopic Examination.—Bladder normal. Ureters catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed:

	Leucocytes per cu. mm	Culture
Bladder	0	Sterile
Right kidney	0	Sterile
Left kidney	0	Sterile

Operation was June 9, 1932, and ethylene anæsthesia was used. The usual left lumbar incision was made. A large solitary cyst about the size of a grapefruit was found springing from the lower pole of the kidney. The cyst was resected and a nephropexy done (Fig. 8).

Result: Uneventful recovery.

Intravenous pyelogram, made July 18, 1933, showed on the left side a slightly distorted pelvis, the right side was normal (Fig. 9).

Solitary Cyst of the Kidney; Stone in the Kidney Pelvis

CASE 7. M. S. G., male, aged 61 years, was admitted to the Presbyterian Hospital, October 22, 1928. His chief complaint was hæmaturia. About 3 months before entering the hospital, patient had noticed that the urine contained blood. He did not experience pain, however, and he thought that the hæmaturia was due to a strain. A roentgen-ray examination at that time showed the presence of a stone in the left kidney. The urine cleared up in about a week and no blood had been noticed until a week ago.

Pain over the left kidney had begun about 6 weeks before and was described as a dull sore ache localized over the left kidney region. The pain did not radiate, it was not severe, and bore no relation to urination, respiration, or bowel movements.

Physical examination was negative. Urinalysis revealed no albumin; no sugar; some blood. Microscopic examination of the sediment revealed a few red blood cells. Blood examination showed red cells, 4,460,000; white blood cells, 7,400; hæmoglobin, 85 per cent. The blood pressure was systolic 104, diastolic 68. The blood Wassermann test revealed a negative reaction. Blood chemistry examination showed urea nitrogen, 23.5; uric acid, 4.4; creatinin, 2.2; non-protein nitrogen, 49.5. Roentgen-ray examination showed the right kidney outline normal. The left kidney outline was normal except for a globular projection at the lower pole which was visible on several films. There was a dense shadow due to a stone in the region of the inferior calyx. A pyelogram which was made of the left side showed it to be normal.

Cystoscopic examination revealed a small median bar. The bladder was otherwise negative. Both ureters were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed:

	Leucocytes per cu. mm.	Cultures	Tubercle bacillus
Bladder	10	Sterile	0
Right kidney	20	Sterile	0
Left kidney	20	Sterile	0

In the thalein test the total output of the dye, was 20 per cent in 30 minutes on the right side; 17.5 per cent in 30 minutes on the left side. Operation on May 8, 1929, was done under ethylene anæsthesia. The stone was removed through a pyelotomy incision, and the cyst (lower pole) was resected.

Result: uneventful recovery.

Blood chemistry, May 10, 1934, revealed creatinin, 1.2; non-protein nitrogen, 36.3.

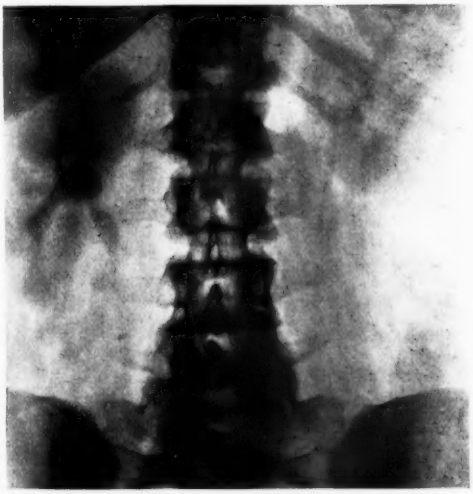


Fig. 9. Intravenous pyelogram after removal of cyst, showing a slightly distorted pelvis. Case 6.

Traumatic Cyst of Lower Pole of the Right Kidney

CASE 8. L. C., male, aged 54 years, was admitted to the Presbyterian Hospital, June 2, 1930. Five months ago, while at work, the patient fell from a height of 6 feet and struck his right side against a roll of paper. He was able to walk, was taken home, and then to a hospital, where he remained for 14 days. He has been unable to work since the injury. Following the fall, blood in the urine was passed, and he had pain of a severe nature in the right upper quadrant which was aggravated by movement. The pain was still present, but not so severe as it was immediately after the injury.

Physical examination showed a marked right costovertebral tenderness and slight rigidity of the right rectus muscle. Urinalysis revealed no sugar; no albumin; blood ++. Microscopic examination of the sediment showed many red blood cells and no casts.

Examination of blood showed red cells, 5,300,000; white cells, 8,750; hæmoglobin, 82 per cent. Blood chemistry showed urea nitrogen, 20; uric acid, 4.6; creatinin, 1.3; non-protein nitrogen, 45.5. The Wassermann and Kahn tests were negative. The roentgen-ray examination showed no evidence of stone in the urinary tract. Intravenous pyelograms were made and that of the right side showed a filling defect compatible with tumor. The left pyelogram was normal. Cystoscopic examination revealed the bladder and ureteral orifices, normal. Examination showed that the urine from the right and left kidneys and from the bladder was free of pus and sterile upon culture. The findings in the intravenous pyelogram were verified by a retrograde pyelogram.

Pre-operative diagnosis: Because of the presence of a filling defect on the right side, as shown both with an intravenous and a retrograde pyelogram, a diagnosis of tumor—probably hypernephroma—was made and operation was advised.

Operation was done June 18, 1930, and ethylene anæsthesia was used. The usual oblique kidney incision was used. Isolation of the kidney was extremely difficult on account of dense adhesions. A cyst the size of a large orange

was found springing from the lower pole of the kidney. The cyst together with a small amount of kidney tissue was resected.

Result: uneventful recovery.

Subsequent course: A right pyelogram was made on April 21, 1931, and this showed a rather small but otherwise normal kidney pelvis.

Adenoma of the Kidney

CASE 9. Miss F. M., aged 24 years. In June, 1920, patient noticed an enlargement on the right side of the abdomen but did not consider it of sufficient importance to consult a physician. Three or 4 months later she called the attention of her family physician to the swelling because of its size. No pain or discomfort or any systemic disturbance occurred until within the last few weeks when there were transient, slight aching pains and discomfort on the right side. The mass, according to her statement, progressively increased in size and at no time were large quantities of urine passed.

Physical examination, March 25, 1924, disclosed a large firm tumor mass which occupied almost the entire right half of the abdomen, extending from about 2 inches above the pelvic brim to the costal arch and medially to the midline. The mass was globular in shape, moved with respiration, was not painful upon palpation, and was about 10 inches in diameter. There was present no evidence of fluctuation.

Blood examination revealed red blood cells 4,000,000; leucocytes, 6,700; hæmoglobin, 95 per cent. The blood pressure was systolic 115, diastolic 75. Urinalysis showed urine cloudy, acid reaction, albumin +, some blood cells, pus +++, no sugar, no casts, some epithelial cells. Roentgen-ray examination showed, in the right kidney region, an area covered by a large soft parts shadow which extended from the eleventh rib to the crest of the ilium and suggested a much enlarged kidney shadow. Cystoscopic examination showed the bladder and ureteral orifices normal. Both ureters were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed:

	Leucocytes per cu. mm.	Urea	Culture	Casts	Red blood cells
Bladder	50		Sterile	0	++
Right kidney	90	1.05%	Sterile	0	++++
Left kidney	70	.87%	Sterile	0	0

Thalein Test.	Right Per cent	Left Per cent
1st 15 minutes	5	12
2nd 30 minutes	8	18
Total: 45 minutes	13	30

Right pyelogram showed slight clubbing of the calyces. In the lying position, the pelvis was opposite the first lumbar vertebra; in standing position, the pelvis was opposite the fourth lumbar vertebra.

Diagnosis: tumor of the right kidney.

Operation was done March 28, 1934, under ethylene anæsthesia, and consisted in resection of the tumor and nephropexy.

Result: uneventful convalescence.

Histological examination revealed an adenoma of the kidney.

Carbuncle of the Kidney

CASE 10. J. R., male, aged eight years was first admitted to the Presbyterian Hospital, September 17, 1932. At this time a diagnosis of acute pyelitis, due to the Staphylococcus aureus was made. The patient was treated with pelvic lavage (1 per cent solution of silver nitrate) and was discharged cured on November 3, 1932. He was admitted to the hospital a second time, February 21, 1933, with chills and fever. There was also noted a mass in the left flank, leucocytosis, and some frequency of urination. A diagnosis of an acute perinephritic abscess was made. The abscess was drained on March 1, 1933, and the patient was discharged on April 18, 1933.

He was admitted to the hospital, a third time, May 31, 1933, because of a persistent sinus in the left flank, tenderness on palpation, and a daily rise of temperature (103 degrees). A retrograde pyelogram (Fig. 10) disclosed a streak of the dye in the region of the left superior calyx. The kidney pelvis and calyces were obscured except for this streak which seemed to be curved upward by a mass which involved the lower two-thirds of the kidney. Urinalysis showed nothing abnormal. The leucocytes numbered 24,700.

Operation was done June 5, 1933, and ethylene and ether anæsthesia was used. The lower half of the left kidney was resected. Result: uneventful convalescence.

Patient was again admitted to the hospital August 10, 1933. Since leaving the hospital about 6 weeks before, he has had no pain, but 10 days ago he began running an afternoon temperature of over 100 degrees, and it was for this reason that he again entered the hospital.

Physical examination revealed extreme tenderness and rigidity to deep palpation on the right side of the abdomen. The impression conveyed was that of the presence of a rounded mass deeply situated in the right upper portion of the abdomen. No tenderness was noted in the left half of the abdomen.

An intravenous pyelogram showed a progressive filling of the right kidney pelvis and calyces—very little at first, but well filled and dilated on the last film. The pyelogram outline on the right extended through the width of three vertebræ. The left showed a small amount of media opposite the first lumbar vertebra and in the ureter. The kidney pelvis and calyces were not outlined and they seemed distorted.

Operation was done September 26, 1933, with ethylene and ether anæsthesia An incision was made over the right kidney and a perirenal abscess was opened and drained. Patient was discharged from the hospital November 3, 1933.

He was admitted a fifth time November 20, 1933. Eight days after leaving the hospital, a daily elevation in temperature recurred up to 101 degrees, but there was no pain. Physical examination was negative except for the right loin wound which had considerable infected granulation tissue with some purulent discharge. No tenderness was noted in the right flank.

Operation was done November 25, 1933, under ethylene anæsthesia. A large amount of granulation tissue was removed with a curette from the site of the old incision in the right kidney. In the upper end of the wound a large deep cavity was curetted.

Because of the persistent rise in temperature, it was deemed advisable to do another operation on the right kidney; therefore, on January 3, 1934, under ethylene anæsthesia, an incision was made over the draining wound and multiple abscesses in the kidney were incised and drained.

Result: Patient made an uneventful recovery and was discharged, March 2, 1934.

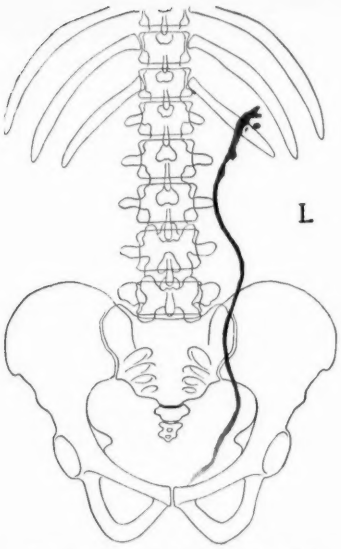


Fig. 10. Retrograde pyelogram showing deformity due to a carbuncle of the lower pole of the kidney. Case 10.

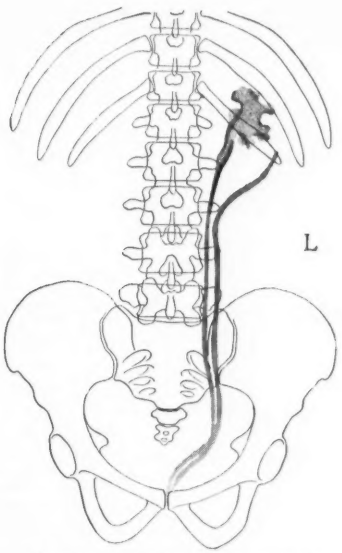


Fig. 11. Showing moderate dilatation of the pelvis of the upper half of the kidney. Case 11.

GROUP II.—DOUBLE KIDNEYS, RESECTION

Complete Reduplication of the Pelvis and Ureter (Left); Infected Hydronephrosis (Upper Half of Left Kidney); and Hydro-Ureter.

CASE 11. Mrs. A. R., aged 50 years, first entered the Presbyterian Hospital, March 2, 1927. Patient gave a history of having had urinary difficulties for 17 years. In 1918 she had had an appendectomy with "no relief" of the symptoms, and within the next 4 years two operations were performed on the bladder for the removal of stones. She was free of symptoms for about 2 years. Upon admission to the hospital, she complained of pain when the bladder was full, pain across the back and left side, frequency of urination, nocturia, urgency, and cloudy urine.

Physical examination revealed tenderness over the left upper quadrant and bladder region; otherwise no abnormal symptoms were noted. Urinalysis disclosed no sugar, no albumin, no blood, pus +++, 1 hyaline cast. Examination of blood revealed: red cells, 4,800,000; leucocytes, 12,300; hæmoglobin, 90 per cent. The blood chemistry examination showed urea nitrogen, 16.86; uric acid, 4.44; creatinin, 1.28, non-protein nitrogen, 39.8. The blood pressure was systolic 200, diastolic 120. The Wassermann test gave a negative reaction. The roentgen-ray examination was negative for stone. Cystoscopic examination showed a normal bladder, with two ureteral orifices on left side; the upper, œdematous. The right ureteral orifice was normal. Both ureters on the left side were catheterized without difficulty or obstruction. The urine at this time showed:

	Leucocytes per cu. mm.	Culture	Tubercle bacillus
Bladder	115	Staphylococcus epidermidis	0
Upper left ureter	586	Staphylococcus epidermidis	0
Lower left ureter	40	Sterile	0

In the thalein test (mixed), the total output of dye was 75 per cent in 1 hour. The left pyelogram showed two pelves and two ureters that terminated in the bladder. In the upper pelvis and its corresponding ureter, dilatation was marked. The lower pelvis and its ureter was normal (Fig. 11). The right pyelogram was normal. Operation was advised and refused.

On second admission, 5 years later, patient had the same symptoms and findings except for marked changes in the pyelogram (Fig. 12).

Operation was done March 4, 1932, ethylene anæsthesia being used. A heminephrectomy of the upper half of the double kidney was performed. The ureter to the upper kidney was removed down to the brim of the pelvis, and then nephropexy of the remaining portion of the kidney. At the upper third of the kidney was a line of constriction, above which the kidney showed marked dilatation and was soft and flaccid. There was one blood supply running into the lower half and one into the upper half.

Result: uneventful recovery.

Bilateral Double Kidney and Ureter; Hydronephrosis of the Upper Half of the Left Kidney Containing Stones; Stone in the Lower Half of Left Kidney

CASE 12. Dr. J. E., aged 51 years, admitted to the Presbyterian Hospital, October 2, 1933. Symptoms began 18 years ago with an attack of left renal colic, 4 months after which a stone was passed. Nine years later he was seized with an attack of severe renal colic on the left side. Dilatations of the ureter and injections of oil were not successful in securing the passage of the stone, so the ureteral orifice was slit and the stone passed later. Patient was symptom free for 8 years when attacks of renal colic recurred on the right side and were associated with intermittent hæmaturia and pyuria. About 1 month later (18 months ago), he passed a stone, since which time the urine has continued to show large quantities of pus. Five weeks before entrance to the hospital patient had severe night sweats with chills,

fever of 102 degrees, and hæmaturia. Ten days ago he experienced a sharp renal colic on the left side which was of short duration and associated with hæmaturia.

Physical examination was negative. Urinalysis showed no albumin, no sugar, no blood, leucocytes +++.

Examination of blood disclosed: red cells, 4,320,000; leucocytes, 6,950; hæmoglobin, 95 per cent. Blood chemistry examination revealed creatinin, 1.9; total non-protein nitrogen, 33.3. The blood pressure was systolic 138, diastolic 98.

Cystoscopic examination showed the bladder to be normal. There were two normally placed ureteral openings. Both ureters were catheterized without difficulty or obstruction. The urine obtained at this time showed the following:

	Leucocytes per cu. mm.	Gram smears	Cultures	Tubercle bacillus
Bladder	720	Gram positive cocci	Staphylococcus albus	o
Right kidney	2	No organisms	Sterile	o
Left kidney	120	Occasional gram positive cocci	Staphylococcus albus ++	o

In the thalein test the left side showed a total output of 3.5 per cent of the dye for 1 hour; the right side, 6.5 per cent for 1 hour. Intravenous pyelograms showed a reduplication of the pelvis and ureter both on the right and left sides. The left upper pelvis showed dilatation and destruction of the calyces; the stone shadows were covered by the media. The lower pelvis showed moderate dilatation (Fig. 13).

Operation was done October 10, 1933, ethylene anæsthesia being used. A heminephrectomy was done, resecting the hydronephrotic upper half of the left kidney which contained stones. A pyelotomy of the lower half of the left kidney was done and two stones were removed.

Result: uneventful recovery.

Double Kidney (Right)

Case 13. Mrs. M. C., aged 41 years, was admitted to the Presbyterian Hospital, October 31, 1923. She had had an attack of pain 14 years ago for which she had been given an injection of morphine, and since then has had attacks of pain in the loin occurring at irregular intervals. Associated with this pain, she experienced distress in the bladder and at the external urethral orifice, and had frequency of urination. She also had an attack of hæmaturia which lasted for one day.

Physical examination was negative except that the lower pole of the right kidney was palpable and tender. Urinalysis showed no sugar, no albumin, no blood, pus +, motile bacilli. Examination of blood revealed: red cells, 4,000,000; leucocytes, 12,200; hæmoglobin, 82 per cent. Blood chemistry showed urea, 30.0; uric acid, 3.0; creatinin, 1.15; non-protein nitrogen, 26. The blood Wassermann test gave negative reaction. The blood pressure was systolic 110, diastolic 78. The roentgen-ray examination was negative for stone. Cystoscopic examination of the bladder was negative. The right and left ureters were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed:

	Leucocytes per cu. mm.	Culture
Bladder	o	Colon bacilli
Right kidney	o	Sterile
Left kidney	o	Sterile

Thalein test returned total output of the dye from the right kidney for 30 minutes, 1.5 per cent; from the left kidney, 8 per cent.

The right pyelogram showed reduplication of the kidney pelvis and ureter. The pyelogram on the left showed a normal condition.

Guinea pig test showed the bladder and the right and left kidneys negative for tuberculosis.

Operation was advised but refused.

Three years later patient returned, complaining of severe attacks of pain. There was essential change in findings. Operation was advised, and on June 3, 1926, under ethylene anæsthesia, patient had a resection of the upper half of the double kidney (hydronephrosis).

Result: uneventful convalescence.

Double Kidney and Double Ureter (Left); Hydronephrosis of Upper Half of Kidney and Hydro-Ureter; Ectopic Ureter

Case 14. P. D., female, aged 9 years, was admitted to the Children's Memorial Hospital, September 11, 1930. Urinary dribbling had been present since birth. As a baby, child never had had a dry diaper. It was thought that the child would do better when older and trained, but these expectations were never realized. She urinated frequently and the urine could not be held for long periods of time; between urinations, there was constant dribbling. Condition had become more marked during the past 2 years.

Physical examination was negative except that over the middle of the sacrum in midline dorsally there was a defect in the bone that admitted two fingers. External genitalia were negative except that "just below the external urethral orifice was seen a small opening that discharged urine." A ureteral catheter was inserted into the opening and a prompt flow of urine obtained.

Cystoscopic examination was carried out while the catheter was in place. A normal bladder and two normally located ureteral orifices were found. The ureters were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed the following:

	Leucocytes per cu. mm.	Cultures
Bladder	195	Bacillus pyocyaneus
Accessory ureter	157	Bacillus pyocyaneus
Left ureter	257	Bacillus pyocyaneus
Right ureter	325	Bacillus pyocyaneus

Urinalysis showed albumin ++, blood o, sugar o. Sediment showed pus and red blood cells. Examination of blood showed red cells, 4,060,000; white cells, 10,950; hæmoglobin, 80 per cent. Roentgen-ray examination was negative for stone but showed spina bifida occulta. A left pyelogram, with shadowgraph catheters in place, showed that the ectopic ureter drained the upper half of the double kidney. There was a marked hydronephrosis in the upper half of the double kidney; the lower half was normal. The right pyelogram showed a moderate hydronephrosis.

Operation was done October 2, 1930, ether anæsthesia being used. A heminephrectomy (left) was done with resection of the accessory ureter which was dilated and tortuous.

Result: uneventful recovery.

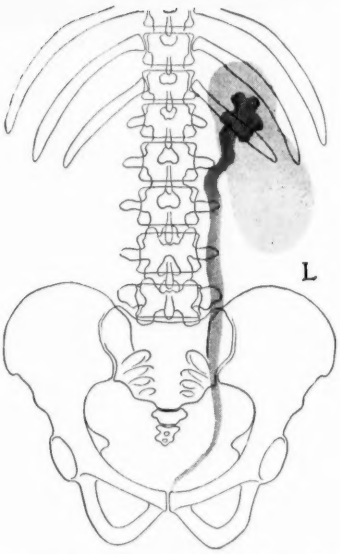


Fig. 12. Showing marked increase in the dilatation of the ureter and pelvis. Case 11.

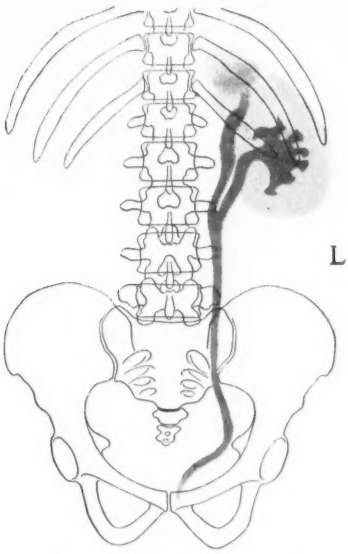


Fig. 13. Dilatation of upper half of double kidney. Moderate of lower pelvis. Stones covered by skiodan. Case 12.

Double Kidney; Hydronephrosis; Hydro-Ureter of Upper Half; Stone in Ureter of Lower Half; Retrograde Movement of the Stone

Case 15. G. H., male, aged 22 years, was admitted to the Presbyterian Hospital, December 29, 1931. He had been in good health up to 5 months ago when he first had an attack of severe pain in the right lumbar region which radiated to the right testicle and was associated with gross blood in the urine. The attacks recurred at intervals of 2 to 3 weeks at first, and during the past few weeks almost were daily with 3 to 4 attacks on some days. The pain sometimes occurred only in the testicle. The hæmaturia was more pronounced during the early attacks than at present.

Physical examination was negative. Urinalysis showed: albumin +; sugar 0; blood ++; sediment, loaded with pus cells; no casts. Cultures showed Bacillus pyocyaneus. Examination of blood showed red cells, 4,310,000; leucocytes, 26,400; hæmoglobin, 80 per cent. The blood Wassermann and Kahn tests gave negative reactions. The blood pressure was systolic 134, diastolic 60. Roentgen-ray examination showed a stone opposite the third lumbar vertebra on the right side. A subsequent roentgen-ray examination showed that the stone had wandered back up into the kidney and was located in the inferior calyx.

Cystoscopic examination revealed a normal bladder. Two normally situated ureteral orifices were seen. They were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed:

	Leucocytes per cu. mm.	Cultures
Bladder	9,600	Bacillus pyocyaneus
Right kidney	80	Bacillus pyocyaneus
Left kidney	40	Sterile

Intravenous pyelograms: on the right side moderate dilatation of the kidney pelvis and clubbing of the calyces were noted. The stone shadow was opposite the third lumbar vertebra. The left side was normal. (The intravenous pyelogram does not show the upper half of the double kidney on the right side. This is due to the fact that the upper half is completely destroyed. There is no secreting tissue present; hence, visualization of the upper half of the double kidney is absent.)

Operation was advised because the ureteral catheterization treatments failed to deliver the stone.

Operation was done March 14, 1932, ethylene and ether anæsthesia being used. Two ureters and a double kidney were found. The upper half of the kidney was completely destroyed and the ureter enormously dilated (Fig. 14). Resection of the upper, hydronephrotic half was carried out (Fig. 15).

A ureterotomy was done with removal of the stone from the ureter that drained the lower half of the double kidney.

Result: uneventful recovery.

Double Kidney and Double Ureter (Right); Hydronephrosis of Lower Half of Kidney; Hydro-Ureter

Case 16. J. P., female, aged 6 years, was admitted to the Children's Memorial Hospital, November 29 1933. She had always been in good health up to 5 months ago when she first developed a dull, aching pain in the right kidney region. The pain was practically constant and at times worse at night, often causing her to cry and keeping her from sleeping. Occasionally she had fever, but there were no urinary symptoms. She has always had enuresis. The pain persisted for 6 weeks before she was taken to a doctor. She had some relief of pain with medication.

Physical examination was negative. Urinalysis showed: albumin +, blood +, sugar 0, pus +, and a few red blood cells. Examination of blood revealed red cells, 4,860,000; leucocytes, 11,300; hæmoglobin, 86 per cent. Blood chemistry showed non-protein nitrogen, 29. The blood pressure was systolic 120, diastolic 80.

Fig. 14. Resected upper half. Case 15.

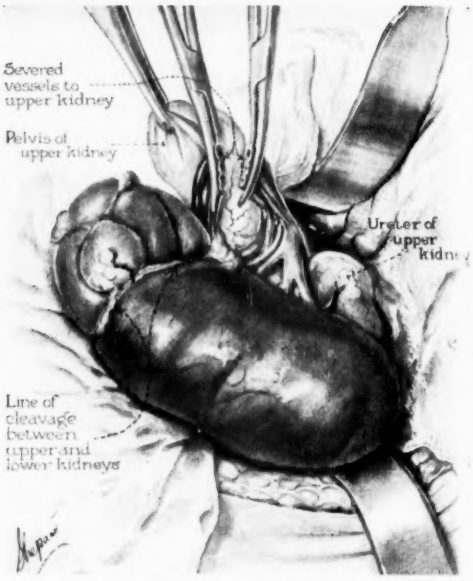

Severed vessels to upper kidney
Pelvis of upper kidney
Ureter of upper kidney
Line of cleavage between upper and lower kidneys

Fig. 15. Note enormous dilatation of upper ureter and pelvis. Case 15.

Roentgen-ray examination was negative for stone. Right pyelogram (intravenous) showed a streak of dye running from the last rib to the upper border of the fourth lumbar vertebra. The left pyelogram was normal. Retrograde pyelogram (made elsewhere) showed much looping of the catheter in the right ureter. The ureter dilated and formed a loop opposite the fourth lumbar. Dilatation of the pelvis and calyces was also noted.

This child developed a pharyngitis and bilateral otitis media and a bilateral paracentesis was done. Further work on the kidney was postponed and patient was discharged, January 10, 1934.

On second admission, February 27, 1934, no essential change in condition was noted.

Operation was done March 1, 1934, ether anæsthesia being used. A heminephrectomy of the lower half of the double kidney was done. The dilated ureter to the lower kidney was removed about 8 centimeters from the pelvis. A nephropexy of the remaining portion of the kidney was done. There was a line of demarcation between the two portions of the kidney, and the lower half was composed of a markedly enlarged hydronephrotic sac with a shell of renal tissue.

Result: uneventful recovery

This case is interesting in that the lower half of the kidney showed the pathological condition and was resected, which is the exception since it is most frequently the upper and embryonic half that is the seat of disease.

My experience agrees with that of Herbst and Polkey who found that the danger of hæmorrhage and fistulæ is rare in the experimental animal.

The question of hæmorrhage was not troublesome in this series of cases, neither at the time of the resection nor subsequently. In the cases of double kidney it was a relatively simple procedure to isolate the vascular supply to the diseased half and to ligate the vessels before the resection was done. If this procedure is followed the operation is practically bloodless.

In the 10 cases of normally formed kidneys in which resection was done, there was no difficulty in controlling the bleeding with sutures. In none of these cases was the bleeding marked. In 1 case with a very slight oozing, a piece of fat was transplanted.

Reference to the possibility of a persistent postoperative urinary fistula has been mentioned by some authors. In this series there were no fistulæ. Careful suturing will, in all probability, prevent the occurrence of a postoperative fistula. When one of the calyces has been opened great care should be exercised in its closure by careful suture.

The determination of the line of resection is generally quite simple. In the cases of double kidney, a line of demarcation between the normal half and the pathological half is readily discernible in most cases, although in a rare case such may be absent or poorly defined.

As a rule, in the cases of solitary cyst but little difficulty is experienced in finding a line of cleavage between the cyst and the kidney. Likewise in the one case of benign tumor reported in this paper, little difficulty was experienced in finding the line of cleavage and it was easily followed by blunt dissection.

If a stone is present and there is also a cavity, no line of cleavage can be demonstrated on the surface of the kidney. In such cases great care was taken to remove these cavities completely.

In the presence of stone two kinds of resections were carried out. In 4 cases the line of incision cut across the kidney from its outer to its inner border. In one case, in which the stone corresponded more or less to a dumb-bell in its configuration, a wedge shaped resection of the kidney was done (Fig. 4).

BIBLIOGRAPHY

1. Bardenheuer, B. Verhandl. deutsch. Gesellsch. f. Chir., 1891, 20: 111.
2. Bloch, O. Brit. M. J., 1896, 2: 1100.
3. Czerny. Quoted by Herczel.
4. Eisendrath, Phifer, and Culver. J. Urol., 1925, 13: 525.
5. Herczel, E. Beitr. z. klin. Chir., 1890, 6: 485.
6. Herbst, R. H., and Polkey, H. J. Surg., Gynec. & Obst., 1931, 51: 213.
7. Judd, E. S. Ann. Surg., 1925, 82: 458.
8. Keetley, C. B. Brit. M. J., 1890, 2: 147.
9. Kummell, H. Deutsche Gesellsch. f. Chir., 1893, 22: 146; Centralbl. f. Chir., 1890, 17: 329.
10. Moynihan, G. A. Brit. M. J., 1902, 1: 263.
11. Peters, W. Beitr. z. klin. Chir., 1923, 129: 716.
12. Rathbun. Am. J. Surg., 1931, 13: 565.
13. Spiegelberg, O. Arch. f. Gynaek., 1870, 1: 146.
14. Waitz, H. Deutsche med. Wchnschr., 1891, 17: 498.
15. Wells, S. Brit. M. J., 1884, 1: 758.
16. Young, H. H. Surg., Gynec. & Obst., 1924, 38: 107.

THROMBOPHLEBITIS AND EMBOLISM

With Special Reference to the Danger of Pulmonary Embolism in the Injection Treatment of Varicose Veins

FRANK V. THEIS, M.D., Chicago, Illinois
From the Department of Surgery, Rush Medical College of the University of Chicago

PULMONARY embolism is generally feared in any case of thrombophlebitis of the lower extremities. Postoperative, infectious, traumatic, or spontaneous thrombophlebitis are too frequently associated with embolism. However, Speed, in 1927, reported 30 deaths from pulmonary embolism within a 10 year period at the Presbyterian Hospital of Chicago with the location of the thrombus in the lower extremities in only 7 cases. The location of the thrombus is an important factor. Thrombophlebitis in the arm veins alone very seldom leads to this complication. Similarly a clinically indistinguishable type of thrombophlebitis produced by the injection of chemical irritants for the obliteration of varicose veins is relatively safe from pulmonary embolism. McPheeters (21, 22), in 1928, after a thorough search of the literature, found only 4 cases of fatal pulmonary embolism in a total of 53,000 cases of varicose veins treated by injection.

In 1931, von Karsten Kettel collected reports of 60,000 treated cases of varicose veins with 10 deaths due to pulmonary embolism. Only 1 of these 10 deaths can be attributed to the present recognized technique. Silverman, in 1931, found reports of 17 deaths besides 1 of his own and 3 non-fatal cases of pulmonary embolism. The almost universal use of chemical irritants for obliteration of varicose veins in itself speaks for the safety of this type of thrombosis.

Doubtless other cases of fatal and non-fatal embolism have occurred and have not been reported. Not only the imminent danger of emboli in other types of thrombophlebitis but the knowledge of unreported cases in obliterating varicose veins has contributed to the hesitancy of the general surgeons in particular in accepting the safety of any case of thrombophlebitis. Since the following case of non-fatal embolism occurred, careful routine physical examinations and selection of cases, strict postinjection instructions, and co-operation of the patient have prevented recurrence of the complication. This case is reported and discussed to point out the dangers which are not usually recognized by the less experienced in this type of procedure.

REPORT OF CASE

Mrs. P., aged 40 years, reported to the surgical dispensary April 7, 1930, suffering with large tortuous varicosities of the left thigh and leg. The internal saphenous vein was markedly enlarged and sacculated. Trendelenburg and Perthes signs were positive. She had had the vein condition for years. She complained of left lower abdominal distress and had lost 12 pounds in weight during the previous month. She attributed this to a recent court experience and worry about her divorce. However, she was referred to the department of general medicine for a complete check up. On April 17, she reported back to the surgical dispensary that she had been examined and nothing was found to interfere with the injection treatment. She was then given one injection of 50 per cent dextrose solution in the left internal saphenous vein at the level of the lower thigh. Two days later the expected moderate inflammatory reaction developed and extended to the fossa ovalis and calf. Consequently she went to bed. Being without a home and having no one to take care of her, she called the social service department and was promptly admitted to the hospital on April 19, 1930.

At the hospital her leg was elevated and hot packs were applied. On the second day her temperature was 100.4 degrees, pulse 84, and respiration 22. On April 22, her general condition was very good and the pain and swelling along the course of the left internal saphenous vein were markedly improved. On April 24, without any change in her general condition, her temperature went up to 102 degrees. She complained of considerable tenderness and distress in the left lower abdomen. No chest pains or dyspnœa were present. April 27 (10 days after injection), she was given magnesium sulphate for constipation. She experienced a sharp chest pain which lasted a few minutes. No cough or dyspnœa was present and physical examination of the chest was negative. Her temperature rose to 103.4 degrees. Additional history then proved that she had had a left lower abdominal abscess 18 years ago which required 10 months hospitalization. Further inquiry revealed that she did not have the required general examination. Because of her recent divorce, she was anxious to have her veins treated without delay.

On April 28, a diagnosis of recurrent extraperitoneal abscess was made. On advice of a gynecologist on May 3 a posterior colpotomy was performed. After thorough exploration only a small amount of clear serous fluid was found. Otherwise conditions were normal. The serous discharge was thought to be a transudate from an abdominal abscess. Colpotomy drainage continued to be practically nil but the temperature fluctuated between 102 and 104 degrees. The left lower abdominal mass rapidly enlarged until it reached the umbilicus. On May 9 the abscess was opened through a left abdominal incision. A large amount of colon odored material was evacuated. There was no question about the abdominal location of the abscess as it was completely walled off by omentum and coils of gut.

The day following operation for draining the abscess a right internal saphenous thrombophlebitis developed. Two

days later the woman experienced sharp pains posteriorly on both sides of the chest. The next day (May 13, 1930) consolidation of the left lower chest was detected. Repeated aspirations of the left chest revealed a thin yellow serous fluid. No organisms were present on culture. Her temperature came down to normal but the abdominal discharge continued for months (discharged to convalescence home December 12, 1930).

After being in fairly good health for more than a year she suddenly developed a panophthalmitis. This seemed to be of embolic origin. The eyeball was removed.

This is an unusually complicated case of pulmonary embolism to be definitely attributed to the injection of dextrose in varicose veins alone. In the first place, the patient had lost 12 pounds in weight and complained of severe left abdominal distress for more than a month prior to her first visit. Undoubtedly, the confinement of the patient to bed, the debility of the patient due to the rapidly developed large abdominal abscess with a very high septic temperature all contributed to the pulmonary embolism. Even if the patient had not received an injection for her varicose veins, postoperative thrombophlebitis and embolism could have developed. In fact, definite evidence of embolism was not detected until after thrombophlebitis developed in the right (opposite) leg, subsequent to the second operation for draining the abdominal abscess.

Several factors may contribute to pulmonary embolism; consequently every means should be used to avoid their occurrence when thrombophlebitis is likely to develop. The following conditions further the development of pulmonary emboli:

1. Limited damage or destruction of the endothelial lining of a vein leading to thrombus formation provides insufficient attachment of the clot to the vein wall.

2. Inactivity of the patient results in the formation of large coagulation stagnation thrombi instead of the limited solid mixed deposition thrombi.

3. Coagulating solutions for intravenous injections produce unattached coagulation clots.

4. Suppurative infection, either caused by direct contamination from surrounding tissues or hæmatogenous in origin is followed by disintegration and loosening of the clot.

5. Direct trauma or massage of the thrombotic area may loosen and detach a portion of the thrombus.

The extent of intimal damage leading to thrombus formation is of primary importance when the danger of emboli is considered (Fig. 1). Pathologists are frequently unable to locate the origin of a fatal embolism in the saphenous or femoral

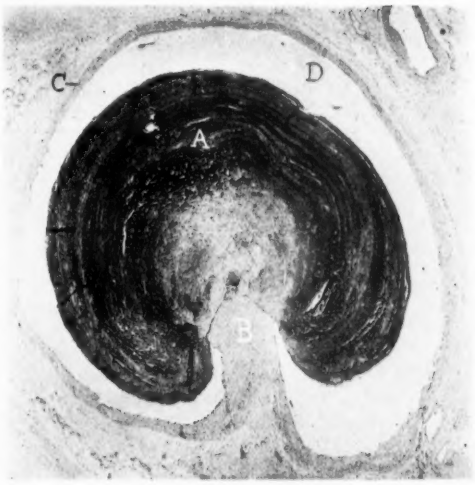


Fig. 1. Photomicrograph of laminated postoperative thrombus of the internal saphenous vein. This specimen was obtained at autopsy following death from postoperative pulmonary embolism. The thrombus, *A*, is attached only at the site of origin on a valve, *B*. The rest of the clot is completely free from any other intimal attachment, *C*. Practically the entire intima is intact, *D*.

vein. However, Aschoff and Kaufmann believe that intimal damage is always present. This damage may be due to trauma at the operative field such as crushing with forceps or ligature, or indirect injury by means of retractors; to infection either hæmatogenous through the vasa vasorum or through direct extension from surrounding tissues; or to chemical and toxic agents. With the injection treatment of varicose veins skilfully administered a non-coagulating chemical irritant is held undiluted in contact with the intima for sufficient time to insure extensive annular destruction of the endothelium (32, 33). The more extensively the intima is destroyed, the more firmly attached will be the thrombus to the vessel wall.

At operation for the removal of biopsy specimens of injected varicose veins, it is apparent that the thrombus is firmly adherent. Forceps and scissors are required to separate the thrombus from the vein or the vein from the surrounding tissues (Fig. 2). I am convinced that the attachment of a deposition thrombus at the site of extensive intima damage prevents its detachment with resulting embolism.

This is contrary to McPheeters' investigation (23) which showed that the reverse flow of blood in varicose veins prevents pulmonary embolism. By means of lipiodal and skiodan injections, roent-

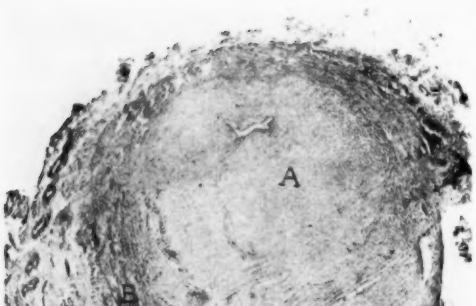


Fig. 2. Photomicrograph of biopsy specimen of thrombosed internal saphenous vein following injection of chemical irritant for the obliteration of varicose veins, *A*. The entire circumference of the intima has been destroyed and no line of demarcation exists between the remaining portion of the vein wall, *B;* obliterating thrombus, *A*, and perivenous tissue, *C*.

genograms revealed the flow of blood away from the heart. Blood pressure readings and posture tests (Trendelenburg and Perthes) substantiated these findings. With the patient in the upright position, the reverse flow of blood in varicose veins would hold true but in the prone position the flow is toward the heart.

Inactivity of the patient subsequent to intimal damage is followed by the formation of a red stagnation clot. Debilitated, bedridden patients are most likely to incur this danger especially in the postoperative period. Red stagnation thrombi are somewhat similar to blood clots *in vitro*, loosely held together, rapidly disintegrating, and contracting away from the wall of the vein. This type of thrombus may occur as the primary clot, i.e., postoperative, or as a secondary coagulation thrombus superimposed on a pre-existing mixed deposition thrombus. These secondary stagnation clots are particularly dangerous in patients confined to bed following the injection treatment of varicose veins.

With the circulation normally maintained by the continued routine activity of the patient, the gradual formation of a deposition thrombus takes place. This is a firm, mixed, and laminated platelet thrombus with a minimum amount of erythrocytes. To each end, proximally and distally, a red clot extends to the next branch (Fig. 3). Under ordinary circulatory conditions, the deposition thrombus at the site of the damaged intima and the proximal and distal red clots are firmly united in a solid mass. However, when thrombosis once starts, the rapidity of the circulating blood determines the size and type of the resulting thrombus. With markedly impaired circulation the additional red thrombi may not stop at the adjacent branch but may extend into the larger and smaller veins. When marked myocardial degeneration exists, unusually large thrombi may develop from the site of very limited endothelial damage.

The greatest danger of emboli exists in the presence of coagulation thrombophlebitis (18). These thrombi may occur either as primary coagulation clots or secondary to a mixed clot as heretofore described. In almost all of the reported and unreported deaths from pulmonary embolism in the injection treatment of varicose veins, confinement of the patient to bed preceded the complication. Perivenous tissue necrosis from the injections, bilateral multiple injections at one sitting, or severe inflammatory reaction from improper selection of cases, or careless surgical asepsis interfere with the normal activity of the patient. Olson reported one death after injection from pulmonary embolism in a patient 3 days after hospitalization. A patient of von Eiselsberg died after 10 days of hospitalization for excision of an area of necrosis and ligation of the saphenous vein. Faure's patient was confined to bed for 16 days after injection; she developed a nonfatal pulmonary embolism the day following her discharge from the hospital. Nobl (12) reported 1 death 3 days after hæmorrhoidectomy in a patient who had received injections 6 weeks previously. Both Isaac and de Takats (7) reported cases in which patients who had received injection treatment developed pulmonary embolism after confinement to bed for emergency appendectomy. McPheeters and Rice had 2 deaths from pulmonary embolism following hospitalization for excision of postinjection necrosis. It is most important that the routine daily activities of the patient should be encouraged during the course of treatments.

The importance of ambulatory treatment of thrombophlebitis when possible cannot be overemphasized. Homans, of the Massachusetts General Hospital, reported 3 deaths from pulmonary embolism in 162 ligation thrombophlebitis cases confined to bed. De Takats (9) advises ambulatory ligations of the internal saphenous vein and has had no deaths from this cause in more than 200 ligations. Vigyazo questions the

advisability of confining patients to bed following any varicose vein operation. Instead of the usual 2 to 3 weeks in bed, he believes it might be best to permit the patient to be up and about the same day. Meyer-Wildisen advises that intravenous or intramuscular injections in general be given in the arms. There is always more movement in the arms than in the legs in bedridden patients. He reports no emboli since this postoperative regimen has been followed. At the Presbyterian Hospital of Chicago 3 bedridden patients developed thrombophlebitis of the arms. No embolic complications occurred.

In my personal series of more than 14,000 injections (882 patients), each one of which was expected to produce an area of thrombophlebitis, only 1 injection was followed by pulmonary emboli and this patient had been confined to bed for 10 days before the complication occurred. Linser, McPheeters (22), and de Takats (8), all advise ambulatory treatment when possible even in the presence of a severe postinjection reaction. However, McPheeters' recommendation that an entire limb be injected at one sitting is entirely too severe to expect the full ambulatory co-operation of the patient. In every case of spontaneous, traumatic, non-suppurative infectious thrombophlebitis in which the patient is already ambulatory, I insist upon continued activity. Even in the presence of a low grade temperature I no longer confine him to bed. In more than 50 such cases I have had no instance of pulmonary embolism or serious complications.

Suppurative infection is extremely serious in the presence of thrombophlebitis. The pyogenic infection might be the cause of the original thrombosis as may occur from a surrounding abscess, from direct inoculation by the injection, or from a septicæmic or pyæmic hæmatogenous dissemination. Disintegration of the accumulated leucocytes produces proteolytic action of the thrombus itself as well as of the attachment of the thrombus to the wall of the vein. Pyæmia and embolism are very commonly observed. This must not be confused with the non-suppurative inflammatory reaction which is often seen. In a series of these cases Brown was unable to get culture growths from either the inflamed vessel or the contained thrombus.

Coagulating chemicals should never be used in intravenous injections. Early in the injection treatment of varicose veins, Pregl's isotonic iodine solution was advocated. Numerous deaths followed the clotting effect of the solution on the blood. With limited attachment of the thrombus on the wall of the vein the extensive coagulation

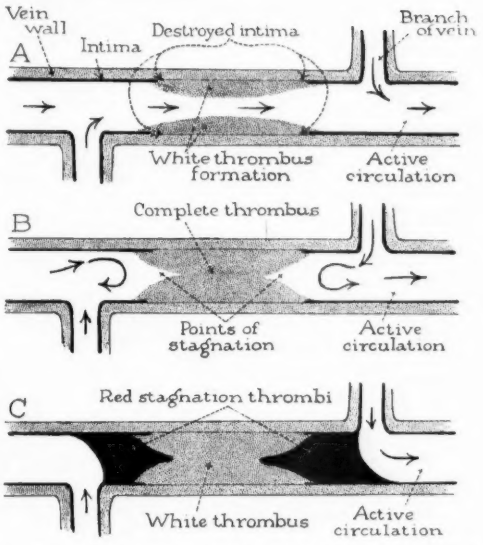


Fig. 3. Schematic illustrations showing the formation of an obliterating thrombus in a blood vessel. With an active circulation the occluding thrombus extends from the site of origin on the damaged intima to the next proximal and distal branches. With an inactive stagnant circulation the thrombus may extend for considerable distance to the larger proximal branches. These latter secondary coagulation thrombi are unattached to the wall of the vein (Fig. 1) and are easily dislodged and broken off to form emboli.

thrombus is easily dislodged. Three of the 10 deaths from pulmonary emboli reported by von Karsten Kettel were due to the use of this solution. Its use has been completely discarded (14).

Direct trauma and massage to a thrombotic area are dangerous. The superficial inflammatory reaction which so frequently accompanies thrombophlebitis is occasionally massaged with a liniment or ointment. Even the more intelligent lay people are often inclined to do this unless definitely informed to the contrary. Direct trauma as in athletics may be the cause of breaking loose of a ragment of the thrombus. Kilbourne reported 1 death following massage of the limb after injection.

PREVENTION OF PULMONARY EMBOLISM AFTER INJECTION

Basically, thrombophlebitis is always a potential source of embolism. The ruthlessness with which emboli strike and the helplessness of treatment compels the use of preventive measures to avoid thrombosis which cause the emboli. Naturally, to produce a thrombophlebitis deliberately could be severely criticized. At the Presbyterian Hospital of Chicago 70 patients were treated dur-

INCIDENCE OF THROMBOPHLEBITIS OF THE LOWER EXTREMITIES AND PULMONARY EMBOLISM AT THE PRESBYTERIAN HOSPITAL OF CHICAGO

Causes	Cases of thrombophlebitis	Pulmonary embolism			
		Frequency		Mortality	
		No.	Percentage	No.	Percentage
Admitted with thrombophlebitis	19*	2	10.5	2‡	100
	2†	2	100	2	100
Postoperative thrombophlebitis	39	8	20.5	2	25
Postpartum thrombophlebitis	10	0	0	0	0
Total number of cases	70	12	17.1	6	50

*Traumatic and spontaneous.
†Suppurative.
‡Death occurred in 1 case following resection of the thrombosed internal saphenous vein.

ing the past 10 years for thrombophlebitis of the lower extremities. Twelve, or 17.1 per cent, developed pulmonary emboli which resulted in 5 deaths—a mortality of 7.1 per cent in patients with thrombophlebitis.

In a series of thirty deaths from postoperative embolism at the Presbyterian Hospital of Chicago, Speed, in 1927, found that the thrombosis occurred only 6 times in the leg and once in the thigh. This indicates that thrombosis in the extremities is less apt to cause embolic death than in other locations. However, Cleland and Barlow report that 2.5 per cent of all autopsies showed pulmonary embolism, and in every instance the original clots were found in the leg veins.

Hunt at the City Hospital of Worcester, Massachusetts, reports 94 surgical, traumatic, and obstetrical cases of thrombophlebitis. Almost 25 per cent of these cases with thrombotic complications died of pulmonary embolism. Tempsky from the Kuettner clinic compiled the frequency of thrombosis and embolism according to the seriousness of the surgical condition and the debility of the patient following operation. In carcinoma of the stomach and rectum, pulmonary emboli occurred in 1 to 3 to 1 to 2 cases of thrombosis, respectively. Simple herniotomy was followed by thrombophlebitis in only 0.8 per cent of the cases, but 25 per cent of these developed fatal pulmonary embolism. In appendectomy one embolism occurred to each 7 of thrombophlebitis. Brown, at the Mayo Clinic, recognized pulmonary infarction in 33 per cent of 87 cases of thrombophlebitis although no deaths from pulmonary embolism occurred.

Frequent suggestions appear in the literature attempting to reduce the incidence of thrombophlebitis with its accompanying danger of embolism. In a statistical review of pulmonary embolism, Henderson emphasized the importance of age, weight, general condition of the patient, efficiency of circulation, bodily inactivity, and infection. Snell found that obesity or weight did not definitely contribute to embolism. Walters, in a symposium on fatal pulmonary embolism before the 1927 Clinical Congress of the American College of Surgeons, expressed the opinion that lowering of the blood pressure, depression of metabolism, and possibly slowing of the circulation as a result of prolonged rest in bed, play an important part in either the predisposition to, or the causation of, postoperative thrombosis and emboli. Following a regimen to overcome these factors including thyroid medication he reports a 75 per cent reduction in fatal pulmonary embolism. Bancroft and Brown believe that changes in the blood itself are probably the most important. On the other hand, Allen, after extensive investigation, states that the "non-specific physiological changes in the blood in all probability play only a minor part in contributing to thrombophlebitis and embolism." Miller believes that the "outstanding single factor concerned with thrombophlebitis and embolism is stasis of the blood in the pelvis." In spite of the investigations and reports with suggestions to reduce the incidence of thrombosis and embolism, many European clinics have found an alarming increase.

In discussing the prevention of postoperative pulmonary embolism, David advises that "patients should not be allowed to remain in one position during the first few days when so little inclination exists to move. The position of the legs should be frequently changed and in almost all abdominal surgery the patient should be turned on the side for certain periods during the day. . . ." It is interesting that in the herein reported 39 cases of postoperative thrombophlebitis of the ex-

tremities at the Presbyterian Hospital of Chicago none occurred on his service. This regimen is particularly valuable in those patients who after injection require confinement to bed for emergency conditions such as appendicitis, etc.

With each injection of a chemical irritant, a deposition mixed thrombus adherent to the damaged vein wall is expected to obliterate the varicose vein permanently. In more than 14,000 injections (882 patients) which I have personally given, an equivalent number of potential sources of emboli is provided. However, with only 1 injection did this complication occur. In this series the patients' ages ranged from 16 years to 89 years with the large majority between 30 and 45 years. At least 8 per cent were obese, 1 per cent cardiac, and ½ per cent controlled diabetics. Another 10 per cent were unusually thin. In 10 per cent of the cases there had been a previous thrombophlebitis. The size of the veins treated varied from the small superficial telangiectatic type to the huge tortuous type covering both leg and thigh. No patient has been refused injection treatment because of age, weight, cardiac compensation, controlled diabetes, or a thrombophlebitis not less than 2 to 5 years previous. However, in less than 4 per cent of the patients, injections were postponed because of recent thrombophlebitis, untreated cardiac condition, diabetes, hyperthyroid, or other systemic infirmities, serious peripheral arterial disease with danger of impending gangrene, local infection, ulcerations, eczema, indurations or swelling of the legs and feet or systemic infections, such as grippe, colds, bronchitis, etc. I have had called to my attention 3 unreported cases of sudden death following the injection treatment. All had been confined to bed but details are lacking as to the terminal clinical picture.

CONCLUSIONS

Pulmonary embolism may occur in any case of thrombophlebitis. In more than 14,000 injections (882 patients) for the obliteration of varicose veins, each one of which was given to produce an area of thrombophlebitis, only 1 was followed by pulmonary embolism and that non-fatal. This did not occur until after 10 days' confinement to bed.

Pulmonary embolism occurs most frequently in bedridden, debilitated, and cardiac patients. With limited attachment of the thrombus to the vessel wall, the deficient circulation of blood aids extensive secondary red stagnation thrombus formation. These are easily dislodged and carried away in the circulation.

Although thrombophlebitis produced by injection of chemical irritants, trauma, or infection is indistinguishable clinically, nevertheless, pathologically there may be a marked difference. The possibility of detachment of a thrombus and embolism formation depends upon the pathological type of thrombus present.

Coagulation or secondary stagnation thrombi because of lack of intimal attachment are particularly prone to cause embolism. The intravenous injection of a coagulating chemical irritant or a very limited break in the intima may be the source of extensive coagulation thrombosis.

Deposition or pulsion (Aschoff) thrombi are expected to obliterate varicose veins following the injection of non-coagulating chemical irritants. Extensive intimal damage provides adequate attachment for the thrombus. There is very little danger of detachment or extensive secondary thrombus formation unless the patient is subsequently bedridden.

Suppurative infection or direct injury to a thrombotic area may contribute to the disintegration or dislodgement of any type of thrombus.

Nevertheless, thrombophlebitis of the extremities irrespective of the cause, is less likely to be followed by pulmonary embolism than thrombosis in other locations.

In the injection treatment of varicose veins proper selection of cases for injection, skilled technique, and complete cooperation of the patient after injection in continuing routine daily activities will lessen the frequency of pulmonary embolism.

BIBLIOGRAPHY

1. Allen, Edgar V. Changes in the blood following operation. Arch. Surg., 1927, 15:254-265.
2. Aschoff, L. Pathological Anatomy. Vol. i. Jena: Gustav Fisher, 1928.
3. Bancroft, Frederick W., and Brown, Stanley. Postoperative thrombosis, thrombophlebitis and embolism. Surg., Gynec. & Obst., 1932, 64:898-906.
4. Brown, George E. Postoperative phlebitis. A clinical study. Arch. Surg., 1927, 15:245-253.
5. Cleland, J. B., and Barlow, D. L. Deaths from pulmonary embolism. M. J. Australia, 1922, 1: 175-176.
6. David, Vernon. Surg., Gynec. & Obst., 1930, 50: 158-159.
7. de Takats, Geza. Causes of failure in injection treatment of varicose veins. J. Am. M. Ass., 1931, 96: 1111–1114.
8. Idem. Resting infection in varicose veins. Its diagnosis and treatment. Am. J. M. Sc., 1932, 184: 57–66.
9. de Takats, Geza, and Quillin, Lawrence. Ligation of saphenous vein. A report of 200 ambulatory operations. Arch. Surg., 1933, 26: 72-88.
10. Faure. Monde méd., 1926, 36:56.
11. Henderson, E. F. Fatal pulmonary embolism. A statistical review. Arch. Surg., 1927, 15:231–236.
12. Hirsch, Maximilian. Is obliterating varicose veins without preliminary high saphenous ligation permissible? Wien. klin. Wchnschr., 1926, 39:1310–1311.

13. HOMANS, JOHN. Nelson System of Surgery. Vol. 3, p. 748. New York: Thomas Nelson & Sons, 1927.
14. HORN, O., and FOGED, J. Risk of embolism in the injection treatment of varices. Clinical and experimental study. Mitt. a.d. Grenzgeb. d. Med. u. Chir., 1931, 42:409.
15. HUNT, E. L. Postoperative thrombosis and embolism. New England J. Med., 1933, 208:730–739.
16. ISAAK, LUDWIG. The danger of emboli in artificial varicose vein sclerosis. Muenchen. med. Wchnschr., 1932, 79:1969.
17. KAUFMANN, EDWARD. Special Pathological Anatomy. Walter de Gruyter and Co., 1931.
18. KETTEL, VON KARSTEN. Fatalities following injection treatment of varices—etiology and pathogenesis of emboli. Zentralbl. f. Chir., 1931, 58:1498–1510.
19. KILBOURNE, NORMAN. Treatment of varicose veins of legs, consideration of safety. J. Am. M. Ass., 1929, 92:1323–1324.
20. LINSER, PAUL. Is the non-operative obliteration of varicose veins dangerous? Zentralbl. f. Gynaek., 1931, 55:1442–1444.
21. MCPHEETERS, H. O. Varicose Veins. Philadelphia: F. A. Davis Company, 1930.
22. MCPHEETERS, H. O., and RICE, C. O. Varicose veins; complications, direct and associated, following injection. J. Am. M. Ass., 1928, 91:1090–1094.
23. MCPHEETERS, H. O., MERKERT, C. E., and LUNDBLAD, ROY A. Mechanics of the reverse flow of blood in varicose veins as proved by blood pressure readings. Surg., Gynec. & Obst., 1932, 55:298–302.
24. MEYER-WILDISEN, ROBERT. Damage from injections and frequency of emboli. Zentralbl. f. Chir., 1932, 59:2263–2266.
25. MILLER, EDWIN M. Surg., Gynec. & Obst., 1930, 50: 157–158.
26. OLSON, O. A. Fatality following varicose vein injection. J. Am. M. Ass., 1927, 89:692.
27. REMENOSKY, E. Quoted by Moszkowicz, L. See note 35.
28. SILVERMAN, ISIDORE. Incidence of embolism in treatment of varicose veins with sclerosing solutions. Report of case. J. Am. M. Ass., 1931, 97: 177–178.
29. SNELL, ALBERT M. Relation of obesity to fatal postoperative pulmonary embolism. Arch. Surg., 1927, 15:237–244.
30. SPEED, KELLOGG. Postoperative pulmonary embolism. Ann. Surg., 1927, 85: 44–53.
31. TEMPSKY, ARTHUR. Postoperative pulmonary embolism. Beitr. z. klin. Chir., 1928, 144:170–177.
32. THEIS, FRANK V. Principles and technic of injection treatment of varicose veins. Surg. Clin. N. America, 1931, 11:157–171.
33. Idem. Basis for recurrence of varices in the various forms of thrombophlebitis. Ann. Surg., 1933, 98: 82.
34. VIGYAZO, JULIUS. Danger of thrombosis and embolism in varicose vein operation. Zentralbl. f. Chir., 1932, 59:2681–2684.
35. VON EISELSBERG, ANTON. Quoted by Moszkowicz, L. Treatment of varicose veins with hypertonic sugar solutions. Wien. med. Wchnschr., 1927, 77:461.
36. WALTERS, WALTMAN. A method of reducing the incidence of fatal postoperative pulmonary embolism. Surg., Gynec. & Obst., 1930, 50:154–157.

EVOLUTION AND PRESENT TECHNIQUE OF GASTROJEJUNOSTOMY

R. W. McNEALY, M.D., F.A.C.S., AND MANUEL E. LICHTENSTEIN, M.D., F.A.C.S., CHICAGO, ILLINOIS
From the Department of Surgery, Northwestern University Medical School and Cook County Hospital

GASTROJEJUNOSTOMY as it is performed today represents the crystallization of 53 years of scientific observation and development. While part of the knowledge of this branch of gastric surgery is the result of laboratory experimentation, probably more has been acquired by the method of trial and error in clinical surgery. The sustained interest in this operation is perhaps occasioned by the indubitable fact that for those conditions in which it is indicated, it is one of the most satisfactory therapeutic procedures known to surgery.

The first 20 years of experience with this operation occurred contemporaneously with the development of abdominal surgery and the establishment of the principles of physiology of the gastro-intestinal tract. These 20 years may be described as the period of "groping in the dark" toward a light which probably even now has not attained its full brilliance. The success of the first gastrojejunostomy suggested that it was an operative procedure of value. Difficulties were encountered early and these were not overcome easily because of the incomplete knowledge of the anatomy and physiology of the gastro-intestinal tract. Some 50 different methods, techniques, and procedures for the operation have been devised. Many of them have been discarded as valueless yet each has made its contribution to the present day method of gastro-intestinal anastomosis. Good results often followed operations which were not technically sound. The words of Kocher (26) in reply to a criticism of his method are worth repeating:

> It is well that Nature is not so ungracious as the surgeon. She allows, just as God allows the sun to shine on good and evil, methods which theoretically are good and bad to be successful.

The first gastrojejunostomy was performed by Anton Woelfler (85) in Billroth's clinic in Vienna on September 28, 1881. Events which led to the performance of this particular operation are of interest. As early as 1874, Gussenbauer and von Winiwarter had performed pylorectomy on dogs successfully. Pean did the first partial gastrectomy in 1879 but his patient died. The first successful partial gastrectomy in man was done by Billroth (3) on February 4, 1881. Woelfler, his assistant, reported 3 additional partial gastric resections later in the same year. With the intention of performing a partial gastric resection on another case, Woelfler opened the abdomen of the patient and found a mass which had infiltrated the surrounding tissues. The infiltration was of such character that gastric resection was impossible. It has been related that he was ready to close the abdomen when Nicoladoni who was present in the clinic at the time, suggested that an anastomosis be made between the stomach and the small intestine. There is no record that even suggests the source of Nicoladoni's inspiration unless it be his acquaintance with the successful performance of entero-anastomosis by Gely and Maisonneuve (36) which was reported in 1864. Woelfler proceeded with the proposal of his colleague and recorded his operative technique as follows:

> The stomach was opened one finger's breadth above the attachment of the gastrocolic ligament at the greater curvature by means of a longitudinal incision 5 centimeters long. A loop of the small intestine was pulled up and split in the same length at the surface situated opposite to the mesentery. The edges of the lumen of the small intestine were then inserted into the edges of the gastric lumen in such a manner that the posterior margins of the intestinal lumen were united with the posterior margins of the lumen of the stomach by internal circular sutures with silk threads. At the anterior margins of the gastric lumen and of the intestinal lumen the margins of the mucous membrane were at first attached to each other, then the serosa and muscularis surfaces by means of a modified Lembert's suture. After careful disinfection of the entire field of operation with carbolic acid, the abdominal layers were again closed, and the united wound was covered with an iodoform dressing. During the entire operation, the stomach and the intestinal loop which had to be inserted, were placed upon disinfected sponges. In order that during the operation no intestinal contents should escape, the intestine was closed off in the efferent and afferent portion with a thick silk thread which passed through the mesentery. This method of temporary closure appears to be the most simple and the most appropriate. The operation was carried out under strictly antiseptic precautions but without the use of a spray. No drainage of the peritoneal cavity was done. The course following the operation was satisfactory in every way. The patient remained completely free from fever and felt progressively better since the day of operation. The annoying vomiting ceased. From day to day the patient could take progressively larger quantities of food. At first only a liquid diet was given. From the eighth day on solid food was given without undue results. The wound of the abdomen healed under a dressing by first intention. Four weeks after the operation, the patient has daily evacuations of stools, which are firm and brown color. By means of the gastroenterostomy the author has excluded the carcinomatous pylorus from the channel of nutrition for which he has found a new way. The food reaching the stomach travels a new way and the inflow of bile and of the pancreatic juice through the afferent portion of the intestinal loop is not prevented. The many physiological questions which are concerned can-

not at present be answered. This much is assured, namely, that all the theoretical considerations which could be cited against the operation are removed by the successful result of the operation.

The final comment of this report is significant:

The success of the operation is established regardless of any theoretical considerations which may be cited against it.

Practically, it is of little importance, even today, whether the good results of the operation are due to the neutralizing action of the intestinal juices on the gastric contents, or to simple drainage facilitated by the new outlet of the stomach.

The fortunate outcome of the first case served as a stimulus to its further use. On October 2, 1881, 4 days after Woelfler's first case, Billroth performed the second gastrojejunostomy (4). His patient also had an irremovable carcinoma of the pylorus which had extended into the pancreas. The operation was terminated within an hour. On the day following the operation, a biliary vomiting developed which persisted until the patient's death on the tenth day. Autopsy showed perfect healing at the site of anastomosis and no peritonitis. The afferent loop of the small intestine measured 11 centimeters. The persistent vomiting was due to a mechanical defect at the site of the anastomosis. A spur formation was produced. The intestine was divided into a larger afferent loop which contained the pancreatic and biliary secretions and a much smaller efferent loop. The lumen of the latter was hidden under the overhanging margin of the gastric stoma. This case pointed out a very important technical error. The afferent loop evacuated its contents into the stomach, while the lumen of the efferent loop was so placed that it could not drain the gastric contents. Vomiting was, therefore, the only means of emptying the stomach of the continuous reflux from the afferent loop. Thus attention was called to the fact that suturing of the viscera could be successfully accomplished but some of the mechanical features of the operation needed further elaboration.

In 1882, Luecke performed successfully the third gastrojejunostomy. The fourth, fifth, and sixth cases were performed in the same year by Lauenstein, Kocher (27), and Rydygier (72), respectively.

In 1883, Woelfler reported 2 modifications of his original operation. These were designed to overcome the difficulties encountered by Billroth in his first gastrojejunostomy. In his first modification he suggested that that part of the lumen which belongs to the afferent branch should be so located that it will be covered for the greater part by gastric wall and that the efferent lumen should communicate with the stomach through an opening having a diameter of 2 to 3 centimeters.

The second modification which he suggested was one in which reliance was placed on constriction of the lumen of the afferent limb by suture. Woelfler's ingenuity was exemplified later in his suggestion of still another operation. He detached the afferent limb from the efferent limb and inserted it a short distance lower down on the efferent limb. This formed a Y-shaped loop anteriorly. Later Roux made use of this principle but performed his operation on the posterior wall of the stomach. In the few operations that had been performed up to this date, no definite rule as to the length of the proximal limb of the intestinal loop was established. It was apparent that if perchance a loop of bowel close to the terminal ileum were applied to the stomach, the major portion of the digestive canal would be excluded from use. It was likewise apparent that if an extremely short loop were stretched around the transverse colon, it might cause obstruction of the colon or become obstructed itself. These considerations led to the performance of the first posterior gastrojejunostomy.

On October 19, 1883, Courvoisier performed a laparotomy for the removal of a pyloric tumor. The mass was irremovably fixed and gastroenterostomy was decided on. The first loop of small intestine which presented itself appeared to be too distant from the stomach. Courvoisier decided to search for the duodenojejunal flexure. The omentum was severed transversely along the greater curvature of the stomach, an opening was made in the transverse mesocolon and the jejunum was found and pulled up. After isolating the site of anastomosis by silk sutures tied over split rubber tubes, one above and one below the isolated loop, an opening 5 centimeters long on the antimesenteric margin of the bowel was made. The stomach was clamped off with a Gussenbauer clamp permitting a fold of the posterior wall of the stomach to remain exposed. This fold was also opened for a distance of 5 centimeters and both lumens were united. The entire operation lasted 2 hours and 50 minutes. The operation apparently was successful. On the eighth postoperative day, the patient made the trip necessary to be presented at a meeting of the Swiss Medical Association at Olten. While there, she became ill, developed a fever and died on the thirteenth postoperative day. The autopsy showed a diffuse phlegmonous infiltration in the right rectus muscle and a diffuse peritonitis. A carcinoma of the pylorus was found. The anastomosis had healed perfectly. The stomach opening was in

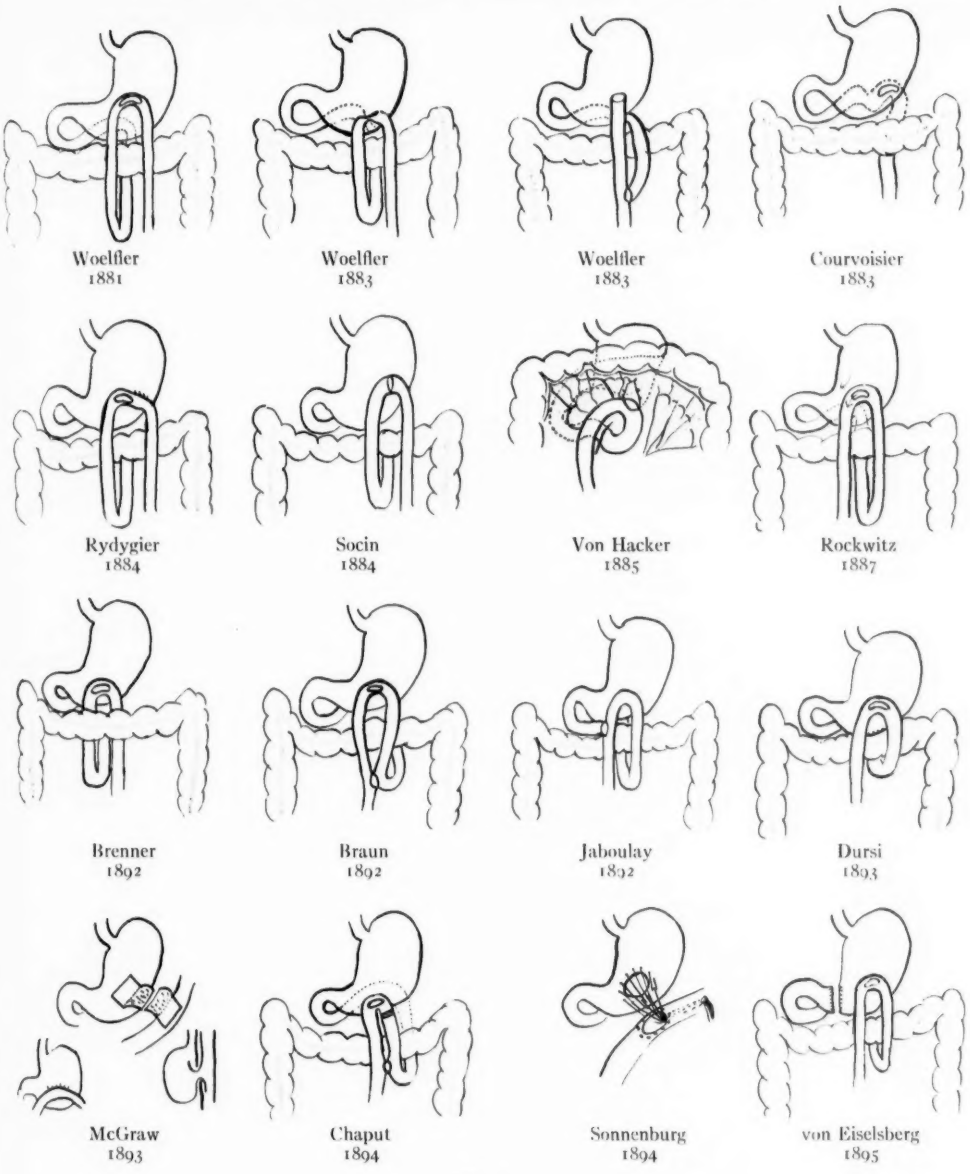


Fig. 1.

the middle of the greater curvature and in the jejunum it was just below the duodenojejunal flexure.

In 1884, Rydygier reported the first gastrojejunostomy performed for pyloric stenosis due to a duodenal ulcer. It is interesting to note that of the 7 gastrojejunostomies thus far reported, only 2 patients survived—one Woelfler's and the other, Luecke's. This operation, therefore, seemed too formidable to suggest its use in the treatment

of a benign lesion. Rydygier's patient was 20 years of age. He had an ulcer of 4 years' duration. The patient was treated conservatively for his vomiting by the use of a stomach tube. Improvement, however, was not satisfactory and it was thought a pyloric resection was needed. At operation, it was found that the ulcer was in the duodenum, and the abdomen was closed. Rydygier thought that if a gastro-enterostomy were done, stagnation would occur in the duodenum. He felt, too, that in view of the high mortality following the operation, it would be better to treat the patient with a stomach tube. Under medical management, the condition continued unabated and the patient lost weight. In desperation another operation was decided on. An anterior gastro-enterostomy was done and the patient made a satisfactory recovery. Later in the same year Rydygier reported 2 additional cases, both successful.

In 1884, Socin of Basel performed a gastro-jejunostomy on a patient who had had a previous pylorectomy. In the same year, Ransohoff performed the first gastro-enterostomy in the United States. By 1885, 18 cases had been published.

On March 22, 1885, Billroth (5) attached the jejunum to the posterior wall of the stomach after passing it through an opening in the transverse mesocolon without perforating the gastrocolic ligament. Although the patient died following this operation, Billroth was not content to discard this method completely but suggested further study of the technical difficulties he had encountered. Von Hacker of Billroth's clinic made anatomical studies on cadavers in an attempt to determine the most favorable site for anastomosing the jejunum to the stomach. A study of the structure of the mesocolon in a number of cadavers revealed several thin avascular regions through which the jejunum could be brought to the posterior gastric wall. Following these studies the operation was performed on patients in the clinic. The results obtained with this new technique were not encouraging. These results were due probably to the nature of the disease—"carcinoma cachexia"—rather than to the technique of the operation.

In 1886, Morse performed the first gastro-enterostomy in England.

In 1887, Rockwitz reported 8 consecutive successful cases and emphasized the importance of selecting suitable cases. With reference to indications, he advised that gastro-enterostomy be employed in cases of stenosis of the pylorus and of the duodenum where a resection could not be carried out. In cases of carcinoma, it was to be employed in the presence of extensive adhesions or glandular metastases, since resection was then contra-indicated. Some of his rules for performing this operation are of special interest. He advised against the separation of adhesions and isolation and manipulations of intestinal loops because he believed that they lead to infection and weaken the patient. He also suggested that any loop might be used for the anastomosis, and referred to Nothnagel's experiment (57) for determining the direction of peristaltic waves.

In 1889, Pozzi performed the first gastro-enterostomy in France (49).

In 1892, Braun reported work which he did in 1884. He performed isoperistaltic application of the jejunum to the stomach as was later recommended by Rockwitz. Lauenstein advised entero-anastomosis if vomiting of bile and pancreatic juice occurred too frequently in the postoperative course of patients who had been subjected to gastro-enterostomy. He, however, suggested that any intestinal loop might be attached to the proximal limb of the jejunum to relieve this condition. This was objected to by Braun because in some instances the terminal ileum was used with very unsatisfactory results. It was Braun's conclusion that when entero-enterostomy was indicated it should be performed between the afferent and efferent limbs of the loop used for the gastro-enterostomy. Jaboulay in 1892 suggested an anastomosis of the distal loop with the third portion of the duodenum so that the pancreatic and biliary juices would be carried away from the stomach.

One of the most troublesome complications of gastro-enterostomy was observed in Billroth's first case. This was the occurrence of a postoperative train of symptoms which have since been designated as "vicious circle" (17). The procedures suggested by Woelfler, Braun, Lauenstein, and Jaboulay were for the purpose of avoiding or relieving this complication. The principle underlying these methods was the prevention of reflux of intestinal contents through the gastro-enterostomy stoma.

In 1897, Roux reported his experience with 50 gastro-enterostomy cases performed as follows: Anterior antecolic, 8 cases; posterior retrocolic, 7 cases; Kocher's valve, 2 cases; retrocolic *en Y*, 29 cases; anterior antecolic *en Y*, 2 cases; anterior retrocolic *en Y*, 1 case; anterior retrocolic, 1 case. His mortality was 30 per cent. In the 29 cases in which he performed the posterior *en Y* operation, the mortality was 20.7 per cent. He preferred the posterior *en Y* operation because the opening was made in the most dependent part of the stomach and there was no spur, kink, or valve. In this operation, there was also no pressure on the colon.

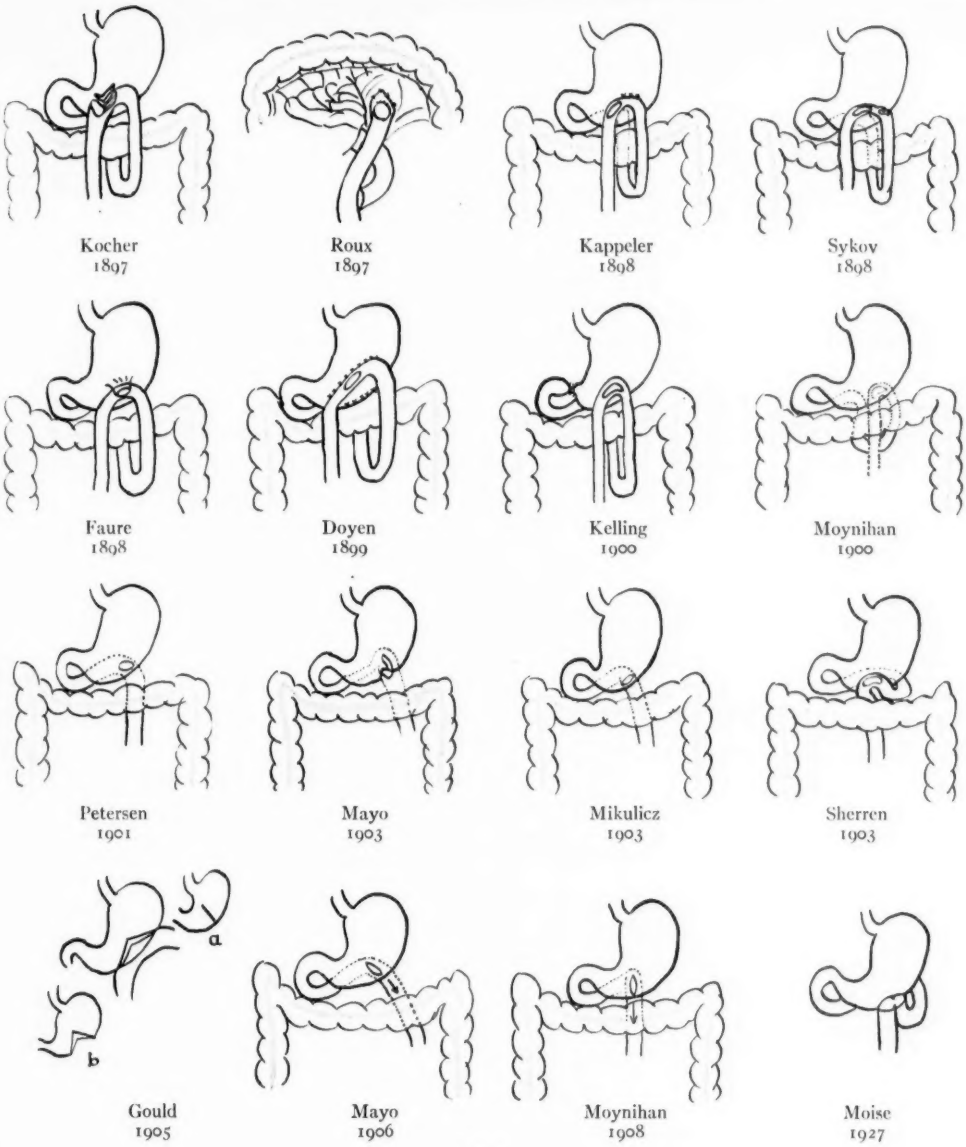


Fig. 2.

The posterior retrocolic operation grew in favor and attracted the attention of many surgeons. It received a further stimulus through the anatomical studies made by Petersen on the relationship between the highest portion of the jejunum and the lowest portion of the stomach. He found that it was possible to anatomose the jejunum with the stomach close to the duodenojejunal flexure without much tension. This obviated the necessity of a proximal loop and made it unnecessary to perform the *en Y* operation which was designed to accomplish the same purpose. In 1901,

Petersen's work was published from Czerny's clinic and its importance was recognized by surgeons throughout the world. It was adopted by the Mayos in this country, by Moynihan in England, and by other large clinics. Aside from some minor modifications, the operation of Petersen is in substance the operation that is performed today.

The interval between 1881 and 1901 saw a group of operations that demonstrated the ingeniousness of the surgeons of that day and the accompanying illustrations portray a number of these interesting procedures. In addition to the mechanical hook-up difficulties experienced, there were the difficulties of avoiding infection and of maintaining the patency of the stomata. The danger of infection in those early days contributed liberally to failure. The incidence of infection was high because the operation was done commonly for malignant pyloric obstruction in the presence of a diminished or absent acidity of the gastric contents. Even today these cases do not fare as well immediately after operation as do those cases in which a benign obstruction is present. To overcome the possibility of infection, operations were devised from time to time in which the anastomosis was performed without opening the mucosa at the time of operation. Thus McGraw (45) in 1891 used an elastic ligature which was designed to cause necrosis eventually of the encompassed tissues and establish an opening between stomach and jejunum. Similarly Postnikow and Podrez used heavy silk ligatures. Souligoux used an angiotribe. Boari and Paul used caustics applied to the mucosa which caused it to slough through at a later date.

There were many failures due to closure of the stoma some time after operation. It was believed that this might be avoided by the use of artificial substances which would splint the mucosa until healing took place and at the same time insure the patency of the opening. Thus Senn described the use of decalcified bone plates in 1888; Robinson advocated raw hide plates in 1890; von Baracz used turnip plates in 1892 and during the same year, Murphy used his famous button. In 1893, Mayo-Robson used the bone bobbin and in 1900 Credé advocated perforated silver plates. Many modifications of each of these methods have been recommended but practically all of these procedures have been discarded by present day surgeons.

The continued use of gastro-enterostomy has stimulated an interest in the anatomy and physiology of that portion of the gastro-intestinal tract related to this operation. Some of the pertinent facts will be reviewed briefly.

ANATOMY AND PHYSIOLOGY

The stomach is a muscular organ divided anatomically into 4 parts: fundus, body, antrum, and pylorus. The latter is not far from its lowest point and lies nearly in the midline of the body. As the stomach distends, it becomes more nearly horizontal by the elongation of the greater curvature. The pylorus passes to the right of the midline and relatively passes above the greater curvature. The gastro-epiploic artery lies at some distance from the greater curvature when the stomach is empty and sends its gastric branches to the anterior and posterior gastric walls. The arrangement of the blood vessels enables rapid distention of the stomach without interference with the blood supply (38). The lesser curvature is more fixed in position and can be divided into 2 parts: the perpendicular portion, which drops nearly vertically from the right margin of the cardiac orifice, somewhat to the left of the midline; and the horizontal or slightly curved portion, which turns sharply to the right and ends at the pylorus. This division of the lesser curvature corresponds to the line of separation between the antrum of the stomach to the right and the body of the stomach to the left.

With these gross external anatomical divisions of the stomach, there are internal anatomical differences. Bisection of the stomach through the greater and lesser curvatures shows that the antrum has a mucosal lining which is rougher, exhibits fewer rugæ, and has a correspondingly lighter (pinker) color than the mucosal lining of the body of the stomach. The thickness of the wall in the antrum is greater and this thickness is due principally to the increased amount of muscle tissue, especially the circular fibers.

These gross anatomical differences are associated with physiological differences. The body of the stomach is the storing and digesting portion of the stomach. Here the greatest activity is shown by the mucosa whose numerous folds afford a large secreting surface. The antrum, conversely, is the active motile and grinding portion of the stomach. Here the greatest activity is exerted by the musculature. In the antrum the peristaltic waves arise approximately 4 times as frequently as they do in the body of the stomach. The difference in the muscular activity of the 2 portions of the stomach accounts also for the marked variations in intragastric pressure. The pressure in the fundus is low as compared to that in the antrum but even here there occur marked fluctuations.

In 1887, von Pfungen measured intragastric pressure on a boy with a gastric fistula. The pressure in the fundus varied only from 5 to 10 millimeters of mercury while in the region of the pylo-

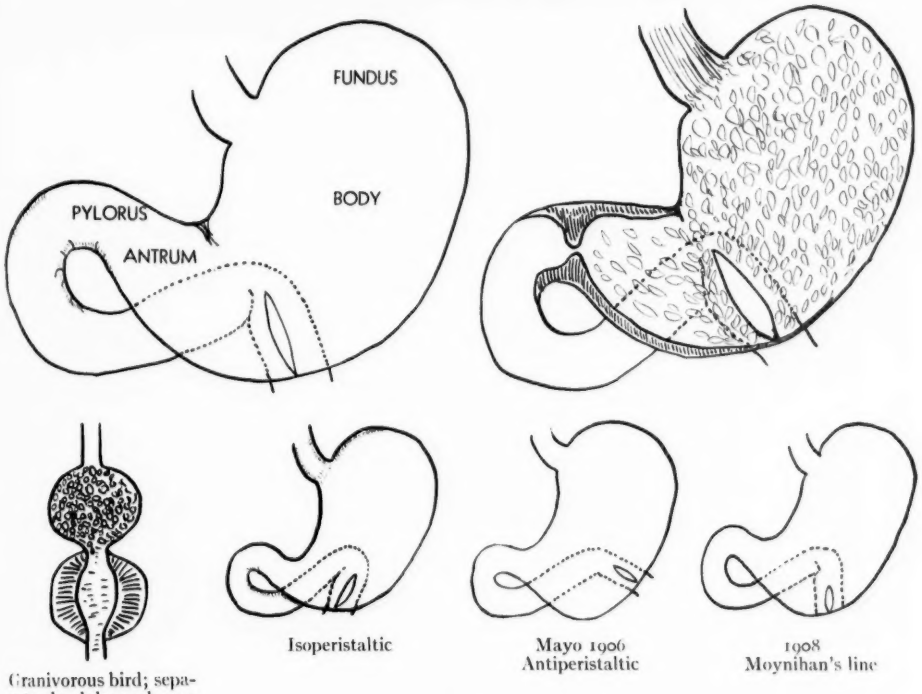


Fig. 3.

rus it varied between 40 and 50 millimeters. Moritz also found that in the fundus the variations of pressure were from 2 to 6 centimeters of water while in the pyloric region the pressure rose to 60 centimeters of water.

The irritability of the antrum is greater than that of the body. Any trauma to the antrum will usually result in spasms of great intensity while this phenomenon is not noted in the body. Ivy has shown that when the antrum is traumatized in making a Pavlov pouch, the intensity of the spasm may be so great that the animals will die of starvation from the spastic obstruction. This does not occur, however, when the surgical procedure is confined to the body of the organ. The distribution of the vagus and sympathetic nerves, those especially associated with muscular activity, is probably greater over the antrum than over the body of the stomach. C. H. Mayo (37) records the fact that X-ray observations have shown that an ulcer that is situated in the antrum will produce more spasm than an ulcer that is located at the pyloric end of the stomach or of the duodenum.

The purpose of this brief exposition of the anatomy and physiology of the stomach is to point out the marked differences that exist between the antrum and the body—differences that are so great that one may consider them functionally as 2 independent organs. In granivorous birds there are present 2 distinct divisions of the stomach (22). One is known as the secretory stomach and the other, the motor stomach (Fig. 3). While these divisions are not so pronounced in mammals, yet in some of the lower forms the divisions are easily recognized. These differences may also account for the conflicting reports in experimental and clinical work. In 1905, Cannon and Blake showed that if the pylorus were patent the gastric contents would not leave through the artificial stoma but would leave by way of the pylorus. Berg made similar observations and reported them in 1913. Their experiments were performed on dogs and cats. It has been repeatedly shown that in humans this is not always true. Numerous X-ray examinations of patients with gastro-enterostomies show that in a high per cent of cases the meal will leave through the gastro-enterostomy stoma

even though the pylorus is patent (58). In many cases the meal will leave by way of both openings. To be sure that the food would leave through the newly formed stoma, Kelling in 1901 and Mayo (42) in 1902 suggested that the pylorus be excluded by plication without division of its continuity. A similar interruption of the pyloric lumen was carried out in 1895 by von Eiselsberg, who, in a case of irremovable pyloric carcinoma, divided the stomach proximal to the tumor and then closed both ends by suture. This procedure was designed not so much to insure the passage of the gastric contents through the new stoma as to prevent the spread of the carcinoma in the direction of the new stoma. In 1914, Hartman showed that the location of the stoma had much to do with the path taken by the meal in leaving the stomach. If the stoma is placed too far away from the pylorus, i.e., far up on the body of the stomach, the new outlet is so remote from the driving portion of the stomach that no sufficient increase in intragastric pressure can possibly influence the movement of food through the new opening. Hartman quotes Tuffier, who states: "I remind you how easy it is to observe on a dog, making use of X-rays, that everything passes through the pylorus, because we know when operating on a dog that the new pylorus can only be placed at a distance of 25 centimeters from the normal pylorus and not less." Those who placed the stoma nearer the pylorus observed different X-ray findings.

Thus we are led to a discussion of the position the stoma should occupy in the operation of gastrojejunostomy. It would seem from the foregoing that the only logical location would be at a point just proximal to the antrum. Thus it would lie outside of the grinding, highly active, irritable portion of the stomach and yet at about the most distal portion of the body of the stomach. This location also would receive the benefit of the greatest intragastric pressure without interfering with the activity of the antrum. The location of the stoma in the antrum is obviously illogical since it acts in itself as a trauma to this highly sensitive portion of the stomach, interferes with its contracting ability, and by its spasm may occlude the lumen of the new stoma. The location of the stoma in the body of the stomach will of course decrease its suitability in direct proportion to its distance proximal to this juxta-antral line.

With the stoma located just proximal to the antrum, the direction would of necessity be vertical, i.e., running from the lesser to the greater curvatures. The line along which the stoma should be placed is described by Moynihan (54) as being in line with the œsophagus. It will be found that when traction is made on several points along the greater curvature, a fold will appear at one point which is in line with the right border of the œsophagus. The stoma should be placed about 1 inch proximal to this line.

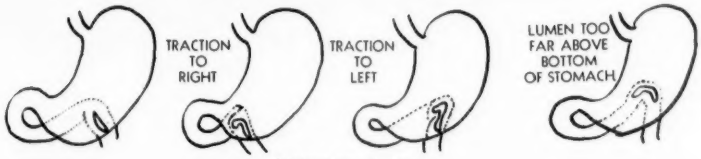
The size of the stoma should, of course, be adequate. Experience has shown that an opening approximately 2½ inches in length in the jejunum and stomach is sufficient for satisfactory evacuation of the stomach.

There are apparently valid objections to having the long axis of the stoma placed in the longitudinal direction of the stomach. Such an opening produces a trough which results in the constant presence of residual gastric contents below the level of the stoma. Incidentally, a longitudinal incision along the greater curvature results in the cutting across of many branches of the gastroepiploic artery at the site of the stoma while a vertical incision runs almost parallel to these vessels. Since drainage is one of the most beneficial results of this operation, the stoma should run down to the greater curvature so that the lowest point of the stoma lies at the lowest point of the stomach. Mayo, in 1905, was so impressed with this detail that he advised carrying the posterior opening past the greater curvature on to the anterior wall of the stomach.

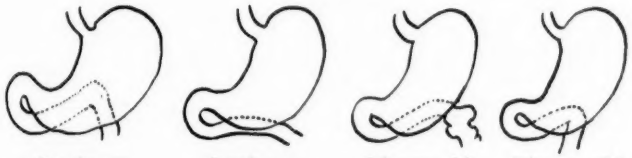
Moynihan early in his work suggested that the opening in the posterior wall of the stomach be inclined downward and to the right (isoperistaltic). Mayo (40), however, advocated that the jejunum be applied obliquely to the left almost at a right angle to Moynihan's line. The basis for this suggestion was that normally the jejunum lies to the left of the vertebral column in a fossa above the left kidney. Further observation led Moynihan (52) to state:

> To say that the jejunum takes a certain line normally from the flexure is probably neither accurate nor reasonable. The ligament of Treitz suspends the jejunum thus allowing it to go to the left or right without kinking. One line is probably as good as another or any line between them as good as either provided no twist is made in the gut. Just as there is no natural direction of the jejunum so there is no best line for the anastomosis.

The location of the stoma in the small intestine should be on the antimesenteric side of the jejunum close to the ligament of Treitz. The anatomical investigations of Petersen in Czerny's clinic at Heidelberg and the recent investigations of MacLeod are of interest because they point out the relationship that exists between the first portion of the jejunum and the posterior wall of the stomach. MacLeod noted that that part of the jejunum which lies below the duodenal flexure as

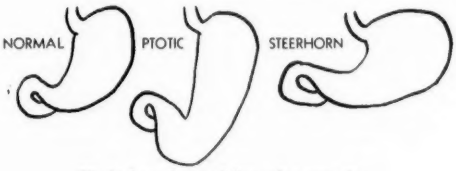


Distortion of stoma

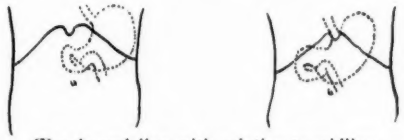


Duodenum directed upward to left | Duodenum directed downward to left | Jejunum with long mesentery | Jejunum with short mesentery

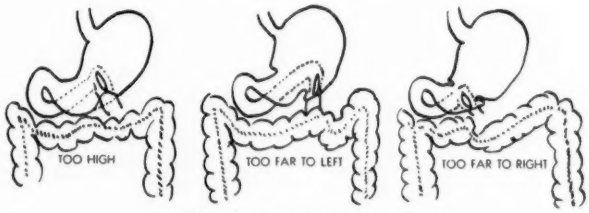
Variations in duodenum and jejunum



Variations in position of stomach



Œsophageal line with relation to midline



Opening in transverse mesocolon

Fig. 4.

it passes either through the transverse mesocolon or immediately between it and the posterior abdominal wall is constant in position regarding its level and lies either in front of or to the left side of the third lumbar vertebra. In some cases of general visceroptosis, it was placed as low as the lower border of the fourth, *but it was usually well above the level of the greater curvature.* The position of the lowest part of the stomach is not constant but lies somewhere on the greater curvature between 2 points, one of which lies 1½ inches proximal to the pylorus and the other 2 inches to the left of the midline. He further demonstrated that by applying traction on the greater curvature, the lowest point of the stomach can be located. This usually lies to the left of the mid-colic artery but

in a few it is to the right. The jejunum, where it crosses the greater curvature, runs sharply downward, in most cases across or just to the right of the most dependent part of the stomach and in a lesser number of cases slightly obliquely to the left. MacLeod found that in every case there is ample room for anastomosis with a satisfactorily large stoma.

Mikulicz also suggested an operation with a very short loop so as to avoid the afferent limb which was responsible for the train of symptoms known as "vicious circle." He demonstrated to the Mayos at Rochester in 1903 a method of application of the jejunum to the stomach that was based on the important observation that the duodenojejunal angle is always above the greater curvature of the stomach and that the jejunum has a vertical course downward where it crosses the greater curvature. In this work Mikulicz used a transverse incision in the jejunum. He effected an anastomosis with the stomach which was satisfactory except for the fact that the transverse opening in the jejunum was too small for adequate drainage of the stomach. This has been abandoned in favor of the longitudinal incision in the jejunum—a procedure which was emphasized by Petersen in 1901.

Occasional variations in the anatomy of the stomach, duodenojejunal angle, and transverse mesocolon, may influence the exact location of the site of anastomosis. Fairchild has emphasized the transverse position of the stomach in a stout individual as contrasted with the longitudinal position in the slender ptotic individual. The jejunum may have either a long or a short mesentery or it may arise (rarely) below the stomach or it may arise at a considerable distance to the left. Thus it seems, the best plan to follow is that suggested by Moynihan and emphasized by Wilkie that the relation between the jejunum and stomach be determined within the abdomen at the time of operation before the anastomosis is made so that no distortion is produced by the anastomosis.

Considerable attention should be given to the selection of the site of the opening in the transverse mesocolon in performing posterior gastrojejunostomy. Within its folds lies the colica media artery whose injury may result in necrosis of the transverse colon. The opening in the transverse mesocolon should be made in an avascular area to the left of this important vessel. In stout individuals the transverse mesocolon may be short, while in slender individuals it may be long. Hence, its relation to the stomach and jejunum should be determined with the organs in the abdomen before anastomosis is attempted. Traction by a short mesocolon may seriously interfere with the functioning of the new stoma. The opening in the mesocolon permits communication between the lesser and greater peritoneal cavities.

Herniation of viscera from the greater sac to the lesser sac may occur if the edges of the opening in the mesocolon are not fixed to the stomach or jejunum. In the early days of this operation, the opening was occasionally sutured to the stomach because it was learned that contraction of the opening about the stoma could occur (43). It was found later that herniation into the lesser peritoneal cavity was common when this suturing was not done. Von Hacker originally sutured the edges of the slit to the stomach and even as late as 1901, Robson and Moynihan suggested that this might be advisable in order to prevent contracture of the slit with consequent narrowing of the jejunum as was noted by Czerny. In 1903, Moynihan (53) had his first experience with herniation into the lesser peritoneal cavity in a case in which he had failed to close the opening in the transverse mesocolon. Subsequently he advocated that this be done in every case. A similar experience by W. J. Mayo (39) resulted in further emphasis on the importance of this procedure. While Moynihan suggested that the slit be sutured to the jejunum, there is a possibility that this may result in obstruction as was recently reported by Torraca. Mayo suggested that the slit be sutured to the site of the anastomosis but in 1912 (39) he reported that in 2 cases where a fat mesocolon was encountered, a constricting band developed which required a second operation for its relief. In his later cases, no difficulties were encountered when the raw edges of the slit were tucked into the lesser peritoneal cavity. This was done by a special application of the suture slightly removed from the raw edge thence through the stomach and jejunum where they lie approximated by suture.

MacArthur offered an important suggestion when he advocated that the slit be sutured to the stomach before the stomach was approximated to the loop of the jejunum for anastomosis. It is obvious that following the anastomosis, the posterior aspect of the gastro-enterostomy is not easily accessible and undue traction may be necessary in order to place the posterior sutures. When clamps are used, he advises that the sutures be placed after the clamp is applied to the stomach and before this clamp is approximated to that on the jejunum.

The use of clamps to facilitate operations on the intestinal canal was described by T. Kocher (28) in 1880. He devised these instruments for intesti-

nal resection but their use for other surgical procedures in intestinal work was made apparent. In 1893, W. A. Lane made use of clamps with curved flat blades that were devised by T. Smith for abdominal operations. By 1895, the use of clamps became well established and they were illustrated in the textbook of surgery by Doyen. In 1900, H. Littlewood of Leeds described the use of rubber covered Doyen clamps in performing gastrojejunostomy. Moynihan early made use of them and it is due probably to his extensive writing that they have become popularized. His demonstration of their use to the Mayos in 1903 at Rochester was the first time they were used in this country. It should be noted, however, that clamps are not considered an absolute necessity and many surgeons prefer to effect the anastomosis without their use. In obese individuals, those with a short transverse mesocolon or a short mesentery of the jejunum, the use of clamps often is made almost impossible.

The type of suture material used in gastrojejunostomy has been a controversial subject for some time. Increasing experience has shown that the use of absorbable material is probably most satisfactory. The presence of non-absorbable suture material which may ulcerate through the wall of the viscus and result in secondary hæmorrhage or chronic ulceration has led to the suggestion that this type of suture be discarded (77). In the experience of many surgeons, there have occurred instances when on reopening the stomach non-absorbable suture materials were found to hang from the site of anastomosis in the lumen of the jejunum. There are some who still prefer the use of silk or linen for all layers of the anastomosis. Others are divided in their selection; some use catgut for the mucosa and linen or silk for the seromuscular layer or vice versa. The use of interrupted non-absorbable sutures to re-enforce the anastomotic sites of particular stress has been rather widely adopted. These are placed as interrupted sutures in the seromuscular layer. More liberal use of these interrupted sutures is made in carcinoma of the stomach or other cachectic states.

The depth of penetration of the suture is of importance. The toughest layer of the intestinal canal is the submucosa and it is into this layer that the seromuscular suture must penetrate if it is to serve its purpose best. It is practically impossible to make this an aseptic suture since the mucosal glands dip into this layer to such an extent that an adequate stitch must of necessity penetrate some of these glands, at least in the jejunum. It has been aptly stated by Sweet that more lives are lost because the suture did not penetrate deeply enough than because of infections resulting from a deep penetration of the suture. The peritoneum may well care for the slight infection engendered by a septic suture but may be powerless against a leak occasioned by the separation of the approximated viscera which occurs as a result of inadequate suturing. It has been found satisfactory to ligate the vessels traversing the submucosa at the site of the stoma in the stomach after the superficial layers have been incised. The larger vessels must be ligated if postoperative hæmorrhage is to be consistently avoided. The lock stitch which usually is employed as a hæmostatic suture, although adequate to control the capillary oozing of the incised mucosa of the stomach and jejunum, is not adequate for hæmostasis of the larger gastric vessels that are severed. This is especially true when clamps are used to fix and approximate the loops of stomach and jejunum for anastomosis. As soon as the clamps are removed and the viscera released from their fixed position of extension, muscular contractions tend to decrease the size of the stoma but the fixed suture now has redundant loops which no longer compress the vessels within their grasp.

It should be noted also that suturing should be for the sole purpose of making adequate approximation of the tissues, thus permitting nature to effect a healing of the approximated structures. When excessive traction is made in placing the sutures, strangulation of the tissues ensues with resulting necrosis and sloughing. This may result not only in secondary hæmorrhage but also in leakage into the peritoneal cavity.

At the time of incision into the stomach and jejunum the free secretions should be removed at once by gentle sponging. These secretions are rich in enzymes, which if allowed to be spread about may favor rapid absorption of the sutures. When the placing of sutures has been completed, it is essential that there be no oozing of blood from the suture line and that all raw surfaces be adequately covered. This will prevent the formation of scar tissue which might constrict or distort the stoma. Before the operation has been concluded, a careful inspection and survey should be made to establish: (1) the correct mechanical arrangement of the anastomosis; (2) the absence of twists or kinks; (3) the patency of the stoma. Before the abdomen is closed the viscera are so arranged that the new stoma hangs free to the left of the vertebral column in order that the site of anastomosis will not be compressed between the anterior abdominal wall and the vertebral bodies. The greater omentum is replaced over the under surface of the anterior abdominal wall and closure of

the abdomen is made by layers without drainage. The posterior sheath of the rectus muscle and peritoneum may be closed by a continuous suture but the anterior layer of the rectus sheath had probably better be closed in most instances with interrupted sutures.

BIBLIOGRAPHY

1. VON BARACZ, R. Turnip plates instead of Senn's bone plates. Arch. f. klin. Chir., 1892, 44:580.
2. BERG, A. A. The influence of gastro-enterostomy on gastric and duodenal ulcers. J. Am. M. Ass., 1913, 60:881.
3. BILLROTH, T. Reports on Billroth's operation. Wien. med. Wchnschr., 1881, 31:595, 634.
4. Idem. Reported by Woelfler, A. (85).
5. Idem. Reported by von Hacker (20).
6. BOARI, A. Two stage operation with caustics. Clin. Chir., 1897, p. 290.
7. BRAUN, H. Gastro-enterostomy and entero-anastomosis carried out simultaneously. Arch. f. klin. Chir., 1892, 45:361.
8. BRENNER, A. Technique of gastro-enterostomy. Wien. klin. Wchnschr., 1892, 5:375.
9. CANNON, W. B., and BLAKE, J. B. Gastro-enterostomy and pyloroplasty. Ann. Surg., 1905, 4:5.
10. CHAPUT. Described in Handbook of Practical Surgery, p. 198, von Bergman, Bruns, Mikulicz. Stuttgart: F. Enke, 1900.
11. COURVOISIER, L. G. Gastro-enterostomy according to Woelfler in case of inoperable cancer of pylorus. Death. Zentralbl. f. Chir., 1883, 10:794.
12. CREDÉ. Gastro-intestinal anastomosis. Zentralbl. f. Chir., 1900, 27:65.
13. DOYEN, E. L. Surgical Treatment of Diseases of Stomach and Duodenum. Paris: Masson & Co., 1895.
14. VON EISELSBERG, A. F. Sidetracking of inoperative pyloric stricture, etc. Arch. f. klin. Chir., 1895, 50:919.
15. FAIRCHILD, F. R. Obstruction following gastro-enterostomy: its mechanical factors. Calif. & West. Med., 1930, 33:860.
16. FAURE, J. L. New method of gastro-enterostomy by means of invagination. Rev. de gynéc. et de chir. abd., 1898, 2:81.
17. FOWLER, G. R. The "vicious circle" following gastro-enterostomy. Ann. Surg., 1902, 36:395.
18. GOULD, A. H. A new method of performing gastro-enterostomy. Boston M. & S. J., 1905, 62:66.
19. GUSSENBAUER, C., and VON WINIWARTER, A. Partial gastric resection. Arch. f. klin. Chir., 1876, 19:347.
20. VON HACKER, V. R. Discussion and statistics on gastric resection and gastro-enterostomy. Arch. f. klin. Chir., 1885, 32:616.
21. HARTMAN, H. Function of the gastro-enterostomy opening in cases of permeable pylorus. Ann. Surg., 1914, 59:832.
22. HUNTINGTON, G. S. The Anatomy of the Human Peritoneum and Abdominal Cavity. Philadelphia: Lee Bros. & Co., 1903.
23. IVY, A. C. Personal communication.
24. JABOULAY, M. Gastro-enterostomy. Jejuno-duodenostomy. Resection of pylorus. Arch. prov. de chir., 1892, 50:1–22.
25. KELLING, G. Surgery of the stomach. Arch. f. klin. Chir., 1900, 62:1.
26. KOCHER, T. Text Book of Operative Surgery, p. 200. New York: Macmillan Company, 1903.
27. Idem. Quoted by Monprofit, A. (49).
28. Idem. On a method of bowel resection, etc. Zentralbl. f. Chir., 1880, 29:465.
29. Idem. Text Book of Surgery. Jena: G. Fischer, 1897.
30. LANE, W. A. Clamp forceps in abdominal operations. Lancet, 1893, 2:813.
31. LAUENSTEIN, C. Verhandl. d. deutsch. Gesellsch. f. Chir., 1883, 12:25.
32. LITTLEWOOD, H. Intestinal suture by means of continuous catgut stitch and excision of mucous membrane. Lancet, 1901, 1:1817.
33. LUECKE, A. Quoted by Woelfler (86).
34. MACARTHUR, A. A. Personal communication.
35. MACLEOD, O. Some anatomical and mechanical factors in posterior gastro-enterostomy. Lancet, 1926, 211:400.
36. MAISONNEUVE, J. G. Clinical Surgery. Vol. 2, p. 439, et seq. Paris, 1864.
37. MAYO, C. H. Discussion on gastric surgery. Ann. Surg., 1929, 90:543.
38. MAYO, W. J. Review of five hundred cases of gastro-enterostomy. Ann. Surg., 1905, 42:21.
39. Idem. Management of opening in transverse mesocolon. Ann. Surg., 1912, 55:398.
40. Idem. Technique of gastrojejunostomy. Ann. Surg., 1906, 43:537.
41. Idem. Duodenal ulcer—a critical review of 58 operated cases with some remarks on gastrojejunostomy. Ann. Surg., 1904, 40:900.
42. Idem. Operations upon the stomach. Ann. Surg., 1903, 38:42.
43. Idem. Complications of gastro-enterostomy. Ann. Surg., 1902, 36:231.
44. MAYO-ROBSON, A. W. The Bone-Bobbin. Brit. M. J., 1893, 1:688.
45. MCGRAW, T. A. Upon the use of elastic ligature in surgery of the intestines. J. Am. M. Ass., 1891, 16:685.
46. Idem. On a method of making intestinal anastomosis which will prevent contraction of the orifice. Ann. Surg., 1893, 18:313.
47. MIKULICZ, J. Quoted by Mayo (38).
48. MOISE, T. S., et al. A method of gastro-enterostomy with a transverse jejunal incision. Surg., Gynec. & Obst., 1927, 44:824.
49. MONPROFIT, A. Gastro-enterostomy. Gaz. med. de Par., 1904, 75:73; 85.
50. MORITZ. Studies on the motor activities of the stomach. Ztschr. f. Biol., 1895, 32:359.
51. MORSE. Quoted by Monprofit, A. (49).
52. MOYNIHAN, B. G. A. Direction of the jejunum in the operation of gastrojejunostomy. Ann. Surg., 1908, 47:481.
53. Idem. Personal communication.
54. MOYNIHAN'S LINE. Quoted by Mayo, W. J. (38).
55. MURPHY, J. B. Cholecysto-entero-anastomosis, etc. Med. Rec., 1892, 42:665.
56. NICOLADONI, C. Quoted by Woelfler, A. (86).
57. NOTHNAGEL, H. Experiments on physiology of the intestines. Arch. f. path. Anat., 1882, 88:1.
58. OUTLAND, J. H. O., SKINNER, E. H., and CLENDENNING, L. Study of the mechanics of the stomach after gastro-enterostomy by means of the X ray. Surg., Gynec. & Obst., 1913, 17:175.
59. PAUL, F. T. New method for gastro-enterostomy. Brit. M. J., 1898, 1:1437.
60. PEAN, J. Removal of tumors of the stomach by gastrectomy. Gaz. d. hôp., 1879, 52:473.

61. PETERSEN, W. Anatomical and surgical studies in gastro-enterostomy. Beitr. z. klin. Chir., 1901, 29:597.
62. VON PFUNGEN. Quoted by Hartman, H. (21).
63. PODREZ, A. Gastro-enterostomy and entero-anastomosis. Arch. f. klin. Chir., 1898, 57:358.
64. POSTNIKOW, P. The two-stage gastro-enterostomy. Zentralbl. f. Chir., 1892, 19:1018.
65. RANSOHOFF, J. Case report. Med. News, 1884, 45:578.
66. ROBINSON, E. B. The rawhide plate. New York M. J., 1890, 52:429.
67. ROBSON and MOYNIHAN. Diseases of the Stomach. New York: Wm. Wood & Co., 1901.
68. ROCKWITZ, C. Gastro-enterostomy. Deutsche Ztschr. f. Chir., 1887, 25:502.
69. ROUX, C. Gastro-enterostomy. Rev. de gynéc. et de chir. abd., 1897, 1:67.
70. RYDYGIER, K. Quoted by Monprofit, A. (49).
71. Idem. The first case of gastro-enterostomy in stenosis of the duodenum as the result of ulcer. Zentralbl. f. Chir., 1884, 11:63.
72. Idem. A case of gastro-enterostomy. Zentralbl. f. Chir., 1883, 10:241.
73. SENN, N. Enterorrhaphy: its history, technique and present status. J. Am. M. Ass., 1893, 21:215.
74. SHERREN, J. CHOYCE—System of Surgery. Vol. 2, p. 409. New York: Funk & Wagnalls, 1905.
75. SOCIN, A. On stomach surgery. Cor.-Bl. f. schweiz. Aerzte, 1884, 14:513.
76. SONNENBURG. Reported by SCHROETER, F. On gastro-enterostomy. Deutsche Ztschr. f. Chir., 1894, 38:296.
77. SORESI, A. L. Secondary ulcers of the stomach and jejunum. Formation of calculi around unabsorbable material used in suturing the intestinal mucosa. Ann. Surg., 1915, 61:328.
78. SOULIGOUX, M. Operation in two stages with angiotribe. Semaine méd., 1896, 16:283.
79. SWEET, J. E. Technique of surgery of gastro-intestinal tract. Surg., Gynec. & Obst., 1927, 44:811.
80. SYKOFF, W. Valvular method for gastro-enterostomy. Arch. f. klin. Chir., 1898, 56:418.
81. TORRACA, L. Incarceration of anastomotic loop in transverse mesocolon. J. Am. M. Ass., 1932, 97:2173.
82. TUFFIER, M. On ulcer of the stomach. Cong. franç. de m d., 1907, 2:137.
83. D'URSI. Gastro-enterostomy for stenosis of pylorus. Zentralbl. f. Chir., 1893, 20:448.
84. WILKIE, D. P. D. Gastric surgery. Edinburgh M. J., 1919, 23:319.
85. WOELFLER, A. Gastro-enterostomy. Zentralbl. f. Chir., 1881, 45:705.
86. Idem. Verhandl. d. deutsch. Gesellsch. f. Chir., 1883, 12:22.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

FRANKLIN H. MARTIN, M.D.....Managing Editor
ALLEN B. KANAVEL, M.D.....Associate Editor
LOYAL DAVIS, M.D.....Associate Editor

DONALD C. BALFOUR, M.D....Associate, Editorial Staff

MAY, 1935

DRAINAGE OF THE PERITONEAL CAVITY AND INTESTINAL OBSTRUCTION

ACUTE intestinal obstruction is a very grave malady. Exclusive of strangulated hernia, the average mortality is around 50 per cent. Adhesions are the largest single factor in bringing about this malady and the adhesions are due chiefly to either infection or drainage material left temporarily in the peritoneal cavity. One of the most difficult decisions that a surgeon is called on to make is to differentiate between paralytic ileus and mechanical obstruction, as a postoperative complication of the acute surgical abdomen, complicated by peritonitis and treated by drainage. Operative procedures for paralytic ileus are usually not only futile but harmful, but to fail to relieve acute mechanical obstruction promptly is to rob the patient of his chance of life. Altogether, the problem is a most harassing one.

Closure of the infected abdomen without drainage is against tradition, custom, and common sense; on the other hand, the placing of drainage material where it comes in contact with loops of small intestine is to invite obstruction, either as an early or late mishap, or to condemn the patient to an uncertain period of partial disability caused by angulation due to adhesions and these adhesions are often the one thing that spoils an otherwise successful operation.

There is experimental and clinical evidence that drains left in the peritoneal cavity are soon sealed off and there is also abundant proof that the peritoneum is a very efficient tissue in taking care of its own difficulties. This is especially true of infections, provided the cause of the contamination is removed and the door closed to further leakage. When gynecologists began to close the abdomen after removal of acutely infected pelvic organs, many surgeons were aghast and it soon became evident that leakage following intestinal suture was more likely to occur if drains came in contact with the suture line.

Drainage material placed in the peritoneal cavity cephalate to the level of the transverse colon is not so likely to cause obstruction, because the jejunum and ileum are usually found caudal to this level. For this reason acute obstruction is not a common complication of drainage following operations on the biliary duct apparatus, the liver, pancreas, duodenum, or stomach.

The solution of the problem, in part, is to place as little drainage material as possible where it is likely to come in contact with coils of small intestine. In draining a walled-off peritoneal effusion the risk of obstruction is not great. To close the abdomen, however, in the presence of peritonitis seemed a fool-

hardy thing to do, but less and less drainage was being used and, being harassed by the imminence of obstruction, the writer, several years ago, began to close the abdomen where early peritonitis was present at operation. My opinion is based on 90 patients with early diffuse peritonitis occurring in about 600 cases of appendicitis. A satisfactory classification of peritonitis from the standpoint of treatment is to divide these cases into three groups.

1. Early peritonitis, whether localized, spreading or diffuse; whether serous, seropurulent, or purulent, irrespective of the infecting organism and without regard to whether the appendix, which is usually at fault, is catarrhal, thrombotic, gangrenous, or perforated. The essential thing in this group is that the peritonitis is an early one; that is, that the gut is still smooth and glistening and not markedly distended or discolored, and that the coils of intestine are not adherent. In this group we carefully remove the appendix, with a minimum of handling, through a McBurney incision, suck out the liquid exudate and close the peritoneum without drainage. We usually drain the subcutaneous fat. We have had no serious mishap in this group.

2. Localized collections of pus within the peritoneal cavity, so called pelvic, appendicular, subhepatic or subphrenic abscess. These walled-off effusions are drained. There were 20 of them and all recovered.

3. Late, diffuse peritonitis with widespread adhesions between the different abdominal viscera surrounding multiple pockets of pus, distended, discolored, and œdematous intestine, a board-like abdomen, rapid pulse, anxious features, dehydration, and disturbance in the acid base balance. These were all drained and all of the fatalities were in this group, 10 deaths in 30 patients.

This classification gives a good working basis and corresponds fairly well with the British manner of indicating the seriousness of acute appendicitis by using the time factor, 1 day, 2 day, 3 day, etc. The peritoneum has a very remarkable defensive mechanism which apparently reaches its maximum efficiency when not disturbed by wide incisions, rough or prolonged handling, or the presence of drainage material, provided the cause of infection is removed and postoperative leakage controlled.

ARTHUR M. SHIPLEY.

CARCINOMA OF THE BLADDER

THE successful treatment of carcinoma is dependent on its early recognition, its situation, its cell differentiation, its accessibility, and on its tendency to metastasize. Carcinoma of the bladder is unique in that all of these factors apply very definitely, yet distinct progress in its management can hardly be said to have occurred for the past several years.

Symptoms of the disease become manifest soon after its inception, but the patient often disregards Nature's warning because of fear or timidity. It is difficult to understand why a person will consult his physician if blood is vomited, but will attribute blood in his urine to a cold, to bad food recently eaten, or to some unusual strain; yet, though it is not diagnostic, bloody urine is one of the earliest signs of carcinoma of the bladder. Statistics in some institutions show that the average duration of symptoms prior to admission for treatment is 2 years. Obviously if this period is not reduced, the number of inoperable malignant lesions will continue to increase. If small lesions amenable to transurethral surgery are disregarded, it is a matter of record that approximately 25 per cent of the major malignant lesions when first seen are inoperable.

Cell differentiation in a vesical carcinoma is a reliable index in estimating the ultimate

prognosis and is an aid in formulating the method of surgical attack. Tumors whose cells are highly differentiated are growing slowly, so that there is not the tendency to invade the wall of the bladder or to extend beyond it. In fact, many of such lesions are papillary or pedunculated and these lend themselves particularly well to transurethral attack. Likewise, some of the more extensive lesions can be treated unless there is some other complicating condition, such as obesity associated with recent coronary thrombosis. In the latter instance I feel that one can remove the growth more completely by the suprapubic or transvesical approach. Furthermore, a more satisfactory investigation of the vesical wall and adjacent mucosa can be made, and the presence or absence of metastasis, although rarely seen with this grade of carcinoma, ascertained. Accordingly, the prognosis can be more wisely estimated.

Tumors whose cells are less differentiated are more malignant and as a rule invade the wall of the bladder early. They tend to ulcerate and produce considerable bleeding, and they frequently are associated with necrosis and infection. Malignant lesions of this type in any hollow viscus are always a serious surgical problem and the prognosis must usually be guarded. The problem immediately becomes more complicated when the lesion involves the natural orifices of associated organs. As an example, a small ulcerating malignant lesion of the pylorus may easily be resected, but it is a much more difficult and serious situation should even a smaller lesion involve the ampulla of Vater. The same is true in the urinary bladder; most unfortunately, however, these ulcerating, highly malignant lesions are often situated around one or both ureteral orifices and the internal sphincter of the bladder. When the malignant lesion involves both ureters it probably is inoperable. Involvement of a ureter immediately brings up the question of conservation of the affected kidney. Reimplantation of the ureter following segmental resection increases the mortality approximately 20 per cent, but this mortality can be reduced by 50 per cent if the ureter is only ligated and dropped back. The latter procedure is applicable only when there is a sound kidney and free ureter on the opposite side. The fate of the kidney with a reimplanted ureter is much debated and offers an excellent field for investigation.

Randall recently observed that a kidney may cease to function if a malignant lesion is situated near the ureteral meatus. The answer to the high mortality following reimplantation may be partly contained in Randall's observation. If this observation is confirmed by others, early diagnosis and treatment should assume increasing importance, since a high percentage of rapidly infiltrating tumors as well as highly differentiated tumors occur in close proximity to the ureteral meatus. In fact, one of the most frequent and vulnerable points of origin is just above the meatus and directly over the intramural portion of the ureter, so that early partial obstruction of the ureter, at least, may occur although the meatus may be free from involvement. The rapidity with which involvement occurs depends largely on the cellular differentiation.

Fortunately, many vesical tumors are essentially highly differentiated, frequently multiple, and pedunculated. Transurethral resection of the tumor and electrocoagulation of the base, or the application of radium, as indicated, are the rule for such lesions. Some of the less differentiated, more invasive tumors may occasionally be adequately handled by the same principle, but the prevailing opinion of urologists in general is that these lesions should be attacked by the transvesical approach, since the tumor is more accessible and its local re-

moval or destruction is therefore more likely to be complete. This is particularly true if the ureter is involved and segmental resection indicated. Tumors involving the dome and posterior wall are also quite accessible and lend themselves readily to resection.

It would be a distinct advantage in formulating the type of treatment were it possible to ascertain clinically the presence or absence of metastasis. If metastasis has occurred, in the majority of cases only palliative treatment is indicated. The frequency of metastasis in carcinoma of the bladder is about the same as for carcinoma elsewhere, and according to Spooner conforms to the plan set forth by Broders, in his classification of epitheliomata, that the less differentiated tumors metastasize early. He has found that the incidence at necropsy of metastasis of vesical carcinomata is 29 per cent, which indicates that it is much more common than is generally believed, which is contrary to the opinion that vesical carcinomata metastasize late.

From the surgical point of view these findings are extremely important, though somewhat discouraging, because rarely can an extensive operation for carcinoma of the bladder be done, with removal of the involved lymph nodes, without a high mortality. Future development in roentgen therapy may offer a more hopeful prognosis. Inability to determine clinically the presence of metastasis is stressed by Spooner. He noted that in 65 per cent of cases in which metastasis was present the condition was regarded as amenable to surgical treatment; when dealing with a large infiltrating lesion, therefore, the foregoing data are suggestive at least that the peritoneal cavity should be opened and careful exploration performed before a radical resection is carried out, which at best is only temporary if lymph nodes are involved. The clinical significance regarding the possibility of metastasis, malignancy of high grade and a large lesion should be emphasized in a case with symptoms present for one year.

The value of a follow-up system in cases of carcinoma of the bladder cannot be overestimated. Recurrence may take place following any method of surgical treatment, but if the recurring lesions are discovered routinely before they produce symptoms, many of them can be destroyed transurethrally and a good result obtained. For instance, in a recent series of 67 cases in which patients had local recurrences and returned regularly at stated intervals for examination, 75 per cent are alive and free from symptoms referable to the bladder and have been for 5 or more years. VIRGIL S. COUNSELLER.

MASTER SURGEONS OF AMERICA

CHARLES BEYLARD GUERARD DE NANCRÈDE

ON December 30, 1847, Charles Beylard Guerard de Nancrède, was born at Philadelphia, Pennsylvania, the son of Thomas Dixie Nancrède, wholesale importer and May Elizabeth Nancrède, née Bull; he died April 12, 1921, at Ann Arbor, Michigan.

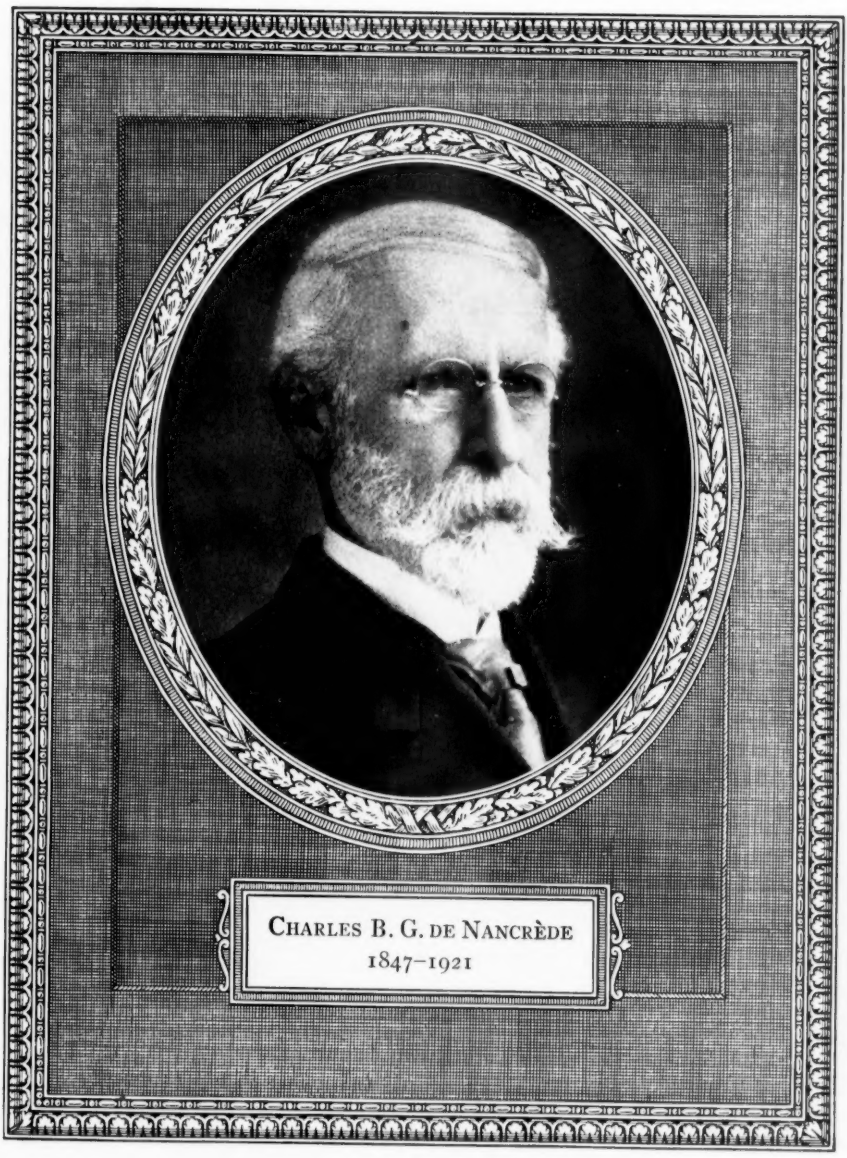
Dr. de Nancrède's paternal ancestors were of French Huguenot origin. His grandfather, Paul Joseph de Nancrède was born in Hericy, France, and came to the United States as a lieutenant in the French Army. He served under Rochambeau and was wounded at Yorktown. He emigrated with his wife to the United States in 1785 and became professor of French at Harvard where he taught for 10 years, from 1787 to 1797.

The early education of Dr. de Nancrède was in private classical schools and in a military school; at the latter he was honored by being selected as one of the cadets who fired the salute at Lincoln's funeral. He later entered the University of Pennsylvania and graduated from the medical department with the degree of doctor of medicine in 1869. Although primarily a medical man and devoted to his work, he had a fondness for art and music. He was particularly interested in painting and once said, "I would have made a better painter in the Fine arts than a surgeon."

His work in physiology, anatomy, and in the eye service at the University of Pennsylvania helped to broaden his medical knowledge. He had considerable training in surgery of the eye and did some major eye surgery, where speed was essential, without the aid of an anæsthetic; this training helped develop his exceptional lightness of touch and speed in surgical technique.

He was one of the first surgeons in Philadelphia to operate for bullet wounds of the stomach and intestine, and later for appendicitis. One of the first operations for the removal of the appendix was done in Philadelphia at the Episcopal Hospital where he was an attending surgeon.

Later he became surgeon and clinical lecturer on rectal and general surgery in the Jefferson Hospital Medical College. He was also attending surgeon at St. Christopher's Hospital for Crippled Children and at the Philadelphia Polyclinic where later he was made emeritus professor of general and orthopedic surgery. He was one of the first surgeons to devote time and study to the diag-



CHARLES B. G. DE NANCRÈDE
1847–1921

UNIV. OF MICH.

nosis and treatment of brain abscess and cortical epilepsy. He was a persistent advocate of Listerism and, with Drs. Keen and Mears, has been more than once accredited before the Academy of Surgery as chiefly responsible for establishing this practice in Philadelphia.

At the time of his coming to Ann Arbor the antiseptic era of surgery had begun. Throughout his early days at Ann Arbor he fought not only for antiseptic surgery but for aseptic surgery as well. We who knew him as students will always remember the emphasis he placed on the necessity of practicing aseptic surgery. In addressing the medical students when he came to Ann Arbor as professor of surgery, it is reported that he said "Gentlemen, we will continue to operate on patients in this hospital. There will probably be many deaths. Where there is a death, do not blame the walls; look elsewhere for the cause."

Prior to accepting the chair of surgery at the University of Michigan he had been striving to enlarge his experience in every branch of the healing art. His untiring interest in other branches of medicine was always impressed upon his students—"I am not a surgeon, but a medical man who operates." His activities were not limited to Ann Arbor, as he held the chair of surgery and of clinical surgery at Dartmouth Medical College from 1900 to 1913, where he gave his course during the summer.

The recognition shown him by his colleagues is indicated by the honors which came to him. He was president of the American Surgical Association and vice president twice; he was major and chief surgeon, United States Volunteers, third division, Second Army Corps during the War with Spain, and served in the Santiago campaign in Cuba with the Fifth Corps. He was made a brevet-lieutenant colonel for services to the wounded under fire, July 1, 1898, and later was commissioned as a major in the Medical Reserve Corps, United States Army, but was not called to active duty in the World War. He was twice president of the Washtenaw County Medical Society; president of the Northern Tri-State Medical Society; corresponding member of the Royal Academy of Medicine, Rome, Italy; member, recorder, and editor of the transactions of the Pathological Society of Philadelphia; member and secretary of the Philadelphia County Medical Society; member of the Pennsylvania State Medical Society, the Ohio State Medical Society, the Colorado State Medical Society, the American Medical Association, the American College of Surgeons, the Detroit Surgical Society, the International Society of Surgery, International Congress at Madrid, Spain, and of the Philadelphia Academy of Natural Science. Aside from the degrees of A.B. and M.D., the following honorary degrees were conferred upon him:—M.A., University of Pennsylvania; M.D. and LL.D., Jefferson Medical School; and A.M. and LL.D., University of Michigan.

Dr. de Nancrède was a contributor to the *International Cyclopedia of Surgery*, the *Cyclopedia of the Diseases of Children*, Burnett's *Cyclopedia of Diseases of the*

Nose and Throat, Dennis' *System of Surgery*, Parks' *System of Surgery* by American authors, the *American Textbook of Surgery*, and Bryant and Buck's *American Practice of Surgery*. He was the author of *Essentials of Anatomy* and of *Principles of Surgery*. The last, a unique work, contained such expressions as "*vis a tergo*," "*pabulum*," "*locus minoris resistentiæ*." The ideas expressed in this book can be studied with great profit today. He published also numerous monographs and papers on anatomical, pathological, and surgical subjects. He carried out research on the nature of the gastric juice of the dog, the effect of blood letting on inflammation, the sterilization of catheters and the rate of absorption of catgut. The personal attention which he gave to his patients no doubt deprived him of sufficient leisure to make his contributions to medicine even more voluminous.

He never spared himself or others where the welfare of the patient was concerned. Night calls had no terrors for him, if the welfare of the patient was concerned. He frequently visited the hospital at night rather than leave this responsibility to his capable assistants. He was, in fact, an individualist.

He was practically ambidextrous and exceedingly deft in his light touch, almost dainty with his small delicate hands. He was very exacting of himself and his staff in the thoroughness of examination of the potential surgical patient by inspection, palpation, auscultation and all the other means available to achieve a diagnosis. Speed in surgical technique combined with thorough anatomical knowledge marked his generation of surgeons and he was an outstanding example.

On June 3, 1872, he was married to Alice Howard Dunnington, of Baltimore, Maryland. Nine children were born to them, and at the time of his death he was survived by his wife and five children. He was an affectionate, indulgent father and very much devoted to his home and family. He had no ambitions along social lines, possibly because he had himself achieved such prominence that he realized the futility of such ambitions.

He was punctilious in his personal appearance, always maintaining an erect military bearing. This latter personal characteristic was undoubtedly developed during his early training and subsequent military duty. Direct in his personal relations with men, he had a high sense of honor and ethics, and a tremendous personal courage. He scorned any chicanery or political maneuvers which tended to develop enemies and he was somewhat contemptuous toward those who failed to agree with his policies. He was opinionated and seemingly somewhat intolerant of the view of others who differed with him, yet he was kindness itself and extremely tolerant of his students and of the younger men on the staff; they respected his knowledge and were very fond of him personally.

The late Dr. Victor C. Vaughan wrote in *A Doctor's Memories* of Dr. de Nancrède; "I cannot overestimate the services rendered to the University by this man." Dr. Reuben Peterson at a meeting of the Washtenaw County Medical

Society, June, 1921, paid a glowing tribute "to the valuable and distinguished services of the great surgeon, Dr. de Nancrède."

One need only mention his high rank as professor of surgery, clinical surgery, and director of the surgical clinics at the University of Michigan from 1889 to 1917, to show that he was universally recognized. His interest in surgery and his devotion to his patients endeared him in the hearts of all who had the privilege of attending the University Medical School during his time. One could not help but be stimulated by his enthusiasm. His influence will long remain in the minds of his students and it may well be said that his life and work represent the true ideal, fulfilled.

GROVER C. PENBERTHY.

EARLY AMERICAN HOSPITALS

THE CHARITY HOSPITAL OF LOUISIANA

A. E. FOSSIER, M.D., New Orleans, Louisiana

ONE hundred and ninety-nine years ago and approximately 18 years after the founding of the City of New Orleans by Bienville, the Charity Hospital of Louisiana was founded. It is the oldest institution of its kind in the United States.

The sailor Jean Louis, a resident of New Orleans, died at noon on the 21st day of January, 1736. He left a holographic will in which the following was specified:

"My debts having been paid and the above provisions having been executed, a sale shall be made of all that remains, which, I bequeath to serve in perpetuity to the founding of a *Hôpital for the Sick of the City of New Orleans*, without any one being able to change my purpose, and to secure the things necessary to succor the sick."

A site was immediately chosen at the extremity of the town, which Governor Miro stated stood upon a portion of the ground allotted to the City's fortification and today corresponds to the square bounded by Rampart, Basin, St. Peter and Toulouse Streets. The house of Madame de Kolly, which formerly was a convent, was bought by Bienville and Salmon. Half of the money of the legacy was expended for beds and the usual hospital equipment, and with the remaining 5,000 livres, a large brick hall was built.

The only description of that hospital handed down to us today is to be had from the building contract, which specified:

Sieur Du Breuil promises, obligates, and binds himself to build, construct and erect on the site of the said hospital a hall measuring forty feet in length by twenty five in breadth, and fourteen in height, including the foundations, the whole in walls of well conditioned bricks, subject to the supervision to plan and payment made, which he promises to construct for the price and sum of two hundred livres per cubic fathom, full or empty, and the other requisites, such as lumber, planks, coverings, iron work, and entire building at the same price as these furnished to His Majesty in this country. The said work was to begin immediately for which he was to be paid on account the sum of three thousand livres in specie.

This contract was signed on the 10th day of June, 1736.

The date of the completion of that building is unknown today but it can be logically stated that its doors were thrown open to the sick poor some time in the fall of 1736. We are told that on March 20, 1737, the hospital had five patients.

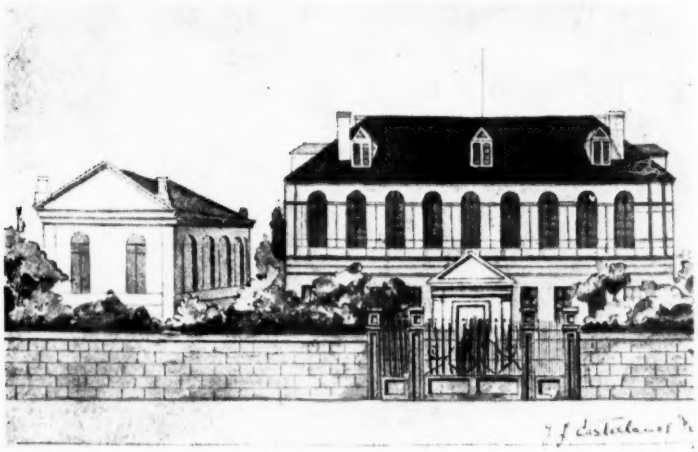
For over forty years this *Hôpital des Pauvres* was a haven of hope for, and administered to, the suffering of those intrepid adventurers who, drawn by the lure of a promised El Dorado and the fallacious inducements held out by the wily John Law, braved privations, hardships, and the pestilences of a primeval country and became stranded on our shores.

The devastating hurricane which played havoc with the city in the summer of the year 1779 converted the Jean Louis Hospital into a heap of ruins; only the kitchen and the storehouse escaped the fury of the storm. The destruction of that hospital resulted in so much consternation and suffering that in speaking of that calamity Governor Don Estavon Miro said: "Many sick paupers are now wandering throughout the city in quest of shelter and succor and are hourly exposed to perish upon the very streets, or in some obscure corner."

Nothing today is known about the medical management of the Jean Louis Hospital. Apparently the professional men of that time played more than a small part in the administration of the charity of that institution. The names of the physicians who administered to those diseased unfortunates during those forty years of its existence are lost to us. There is no record that has escaped the ravages of time and no historian has perpetuated the memory of those altruistic workers in behalf of suffering humanity.

Don Andreas de Almonaster y Roxas, a pecunious old gentleman, who previously had been a war clerk and a civil notary, impelled by the suffering and destitution of the colonists, generously offered not only to rebuild the hospital at his own expense, but to appropriate a yearly sum for its support. He offered the magnificent amount of $114,000.00.

In the year 1782, King Charles II of Spain gave his consent to the rebuilding of this hospital. In that same year, on the same site, and with the



Charity Hospital—1815–1832

salvaged material of the destroyed hospital, a commodious substantial brick edifice rose from the ruins of the original Hospital of Jean Louis. In October, 1786, the rebuilt hospital then called the San Carlos (St. Charles) was dedicated. The following account of that historical occasion is taken from a letter, dated January 17, 1794, by Almonaster addressed to the Governor, Baron de Carondelet. It reads:

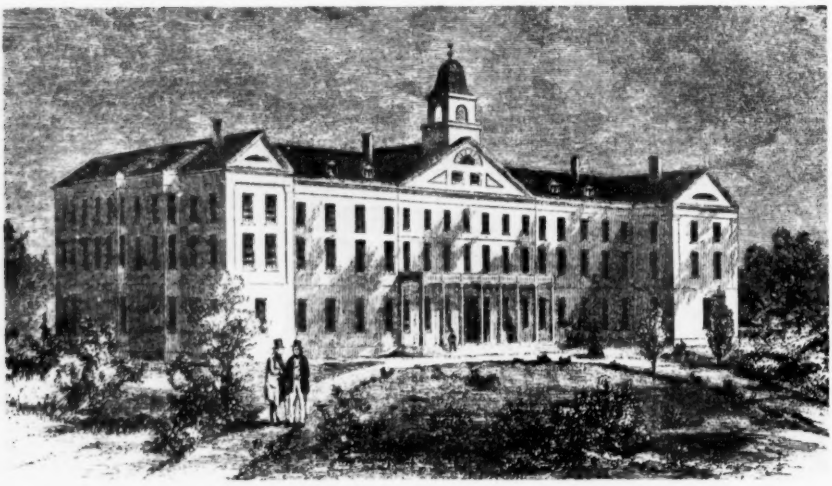
> In October of the year 1786, when the first Mass was said and the sick were received, I was put in possession of the patronage. In conformation to the cited laws, by a solemn act, in the presence of the most distinguished persons of the City, the Lord Governor Don Estabon Miro, your predecessor, and the vicar curate Ecclesiastical Judge Fray Antonio de Sedella, the above named gentlemen bestowed upon me in the name of the King the keys of the said Hospital which he transferred to me by the same act, so that its care may be confined to my zeal and charity.

The first house physicians and surgeons of the Saint Charles hospital were Dr. Don Santiago Leduc, who was appointed to the position in 1792, and Dr. Don Louis Giobellina in 1794.

From an inventory made of the hospital in 1794 we learn that it had 35 small cypress bedsteads, 51 moss mattresses, 30 small linen pillows, 23 large cross-barred muslin mosquito bars, 10 small mosquito bars made of linen, 60 new sheets, 35 white woolen blankets, etc. And an interesting part of this document records as the property of the hospital the following slaves: "One negro carpenter named Petro, aged fifty-five years; one negro carpenter named Joseph, thirty-five years; another Philip, sixty years old; one little negro boy named Andres, of fourteen years; another little negro boy named Francisco, of two and one-half years; one negro girl named Maria, aged eleven; and five lots of ground situated in the city."

This inventory not only gives us today a glimpse of the management of the hospital but reveals many interesting facts. Of special interest to us today is the Chapel. It appears that a large amount of the endowment was expended in its lavish furnishings, which were in striking contrast to the scant equipment for the use of the sick. Drugs were not listed in this document. Remedies must have been procured from the apothecaries. (There were five drug stores at that time in the city). No mention is made of surgical instruments or implements. Without doubt surgery was practiced in that institution. It must be presumed that the operations were performed in the wards on patients in their beds with instruments belonging to the attending physicians.

The census for the year 1796 gave New Orleans 9,756 inhabitants. The Saint Charles was a 24 bed hospital. Its size and capacity in relation to the then population was of more favorable comparison than the hospital of the present day. A concept of the good accomplished, the charity performed, the suffering alleviated, and the number of the sick cared for by that institution can be had only by referring to the vital statistics and the few remaining documents of that time. The extreme suffering, exposure, and the frequent pestilences to which these colonists were continually exposed, and from which only the hardi-



Charity Hospital—1832

est could escape, confirmed the extremely high death rate then current. The total mortality in New Orleans in 1796 was 638, one death to every 13.57 inhabitants, making a ratio of 72.86 per thousand population.

From its foundation the hospital had derived a portion of its income from the legacies of devout and charitable persons. From the earliest time it has been the custom of the French to mention the hospital in their wills. There are scores of testaments extant in which the Charity Hospital is the beneficiary.

Don Almonaster died on the 26th of April, 1798, and he was interred in the Saint Louis Cathedral in New Orleans where his remains still repose.

The San Carlos Hospital and many other public buildings were reduced to ashes by the great conflagration which swept the city on the historical night of the 23rd of September of the year 1809.

A second time the destruction of the hospital resulted in intense suffering and hardship to the indigent sick. The rescued patients were quartered by the Mayor of the City, James Mather for but a day in the upper gallery of the Cabildo, the then City Hall. Temporary quarters were then provided for the patients on the Jourdan plantation, on the site of the present Industrial Canal. This most undesirable location was abandoned after six months of suffering and discomfort to the patients. For five years the Charity Hospital did not have a suitable house for the care of the sick, and it was not until the year 1814 that the square bounded by Canal, Common, Phillipa (now University Place), and Baronne, the present site of the Roosevelt Hotel, was sold by the City to the Administrators of the Charity Hospital. The new hospital was ordered constructed by the Legislature of the State of Louisiana in a bill promulgated April 25, 1811. The corner stone of the edifice was laid in 1815.

The following description of this hospital is taken from the city directory for the year 1823:

> The Charity Hospital, situated No. 147 Canal Street, consists of two large buildings, containing one surgical hall, two large fever wards, one dysentery ward, one ward for chronic cases, one for females, one for convalescents, one bathing room, one apothecary store, and a number of other apartments for the families of the residents, officers, etc. The Hospital has lately undergone a complete repair and reform, and is at this time as clean, wholesome, and well conducted as any institution of the kind in the Union. During the last year, about 1700 sick persons were admitted, 1200 of whom were discharged well, and the remainder died, one-half of which, of Yellow Fever. The lot on which these buildings stand embraces the whole square between Canal, Common, Baronne and Phillipa streets, and is laid off as a garden and poultry yard, etc. About 1300 males and females were admitted during the year 1821, and as many as 130 persons received attention at one time.

It was a 120 bed hospital. This institution in its equipment and management was second to no other in this country. There were bathing facilities, an apothecary, and the fever cases were segregated from surgical cases.

In 1820, an Act of the Legislature provided for the erection of a separate building "to receive and attend such persons as may have fallen into a state of insanity."

Because the rapidly growing population of the city and the extraordinary number of destitute sick from every state of the Union and from every country of the world demanded a larger and more commodious institution, the State Legislature in 1830, authorized the selling of the hospital building on Canal street. In 1832 it was sold to the State for $125,000.00. The main building was then converted into the Capitol of the State of Louisiana. Years afterward the Common street side of the square became the site of the Medical College of Louisiana, now Tulane University.

The present site of the Charity Hospital was selected in 1832. The cost of construction and of the grounds amounted to approximately $150,000.00. The magnificent edifice, the main building of the splendid group of buildings which today comprises the Charity Hospital of Louisiana, and which has reached its centenary, stands today practically the same as on the first day it opened its doors and inaugurated its long service to suffering humanity. Even now it stands as a model of hospital architecture for this climate; its high ceilings, its long and wide halls, its numerous large outside openings and its spacious verandas give it sufficient ventilation, make it cool in summer and at all times cheerful. These are features of construction not usually found in modern institutions.

The following description of the so called "New Charity Hospital" was published in New Orleans in 1840:

> The building was of great size, being about 290 feet in its total length, and three stories high. It is composed of a corps of loges opening into a spacious hall, intersected at right angles by another running lengthwise of the building on which the wards open. From this hall access is had by broad stairs to the upper stories which are similarly divided, and thus to the cupola from which there is a magnificent view of the city and environs. The lower story is occupied by the Library, Physicians' Room, Surgeons' Room, etc., and the second and third stories into wards for the patients, twenty-one in number, as also into four other apartments designed as such. It is calculated to hold 540 patients.
>
> The second floor was appropriated to the use of female patients and was divided as follows: A ward for women of good character, another for those of bad, and also one for the exclusive use of surgical and obstetrical cases. The grounds around it were enclosed with a substantial brick wall, and were handsomely improved and neatly kept.

The portals of this edifice were thrown open to the destitute sick on October 8, 1832.

The Sisters of Charity inaugurated their long period of admirable devotion to the sick and afflicted on January 6, 1834. For over a century they have given uninterrupted altruistic services to the Charity Hospital. Too much cannot be said in praise of these pious religieuses, who spurning all mundane glory, dedicated their lives to unrelenting toil for the alleviation of suffering, and who have braved pestilences and epidemics and even risked existence itself for the devoted love of humanity. The services of the Sisters of Charity are indispensable to the Charity Hospital and their value cannot be appraised merely in monetary terms. The respect they command is an incentive to a greater discipline and a stimulus to more energetic work from their subordinates. In actual work, in enforcing economy, in preventing waste, their services have been invaluable, and their scrupulous management of the domestic and nursing departments has been the leading constituent in the success of the institution through a century of uncertain revenues and inadequate appropriations.

In the past hundred years the growth of the Charity Hospital was rapid. In the 1840's its yearly total number of admissions was from eleven to sixteen thousand patients. It was then one of the largest if not the largest hospital in existence.

Today the Charity Hospital has a capacity of 1,765 beds. The total number of indoor patients for 1931 was 47,182, with a daily average of 2,102. Its outdoor clinics numbered 318,735 consultations. Its records for that year give 26,124 operations including those performed on the eye, ear, nose and throat, and 2,566 obstetrical cases.

A magnificent seven story edifice with a capacity of 250 beds for the convalescents has been constructed.

More than one-third of the deaths registered in New Orleans occur in that institution. Approximately only one-half of the total number of indoor patients are residents of New Orleans, the remainder is composed of individuals from every parish of Louisiana and from every neighboring state. Naturally this unusual condition results in an excessively large non-resident mortality in that city and a corresponding inflation of its death rate. This condition is unique among the large medical centers of this country.

For 100 years this institution has been the cradle of medical education in the South.

From an historical standpoint as well as from the unlimited scope of its charity it stands unique among the great hospitals of the world. It was founded nearly two hundred years ago during the time of the French domination. It was rebuilt, with the sanction of the King of Spain, when Louisiana was a Spanish possession. In the early period of the nineteenth century, its history emblazons in immortal pages an epic of

the greatest distress and suffering ever experienced by man. During the trying days of the Civil War it was converted into a military hospital for the care of the Union soldiers.

In the beginning of the past century when New Orleans was in the making, when it was a city to which unknown thousands of young pioneers, the flower of manhood from every state of the Union and from every country of the world, flocked in quest of wealth or of refuge from political despotism and religious persecution, unfortunately too often to have their hopes and ambitions shattered, to become a prey to the ever recurrent epidemics or to prematurely fill a wet grave, the Charity Hospital was their only haven in their dire hours of suffering and despair.

Then it was not the Charity Hospital of Louisiana, but the Charity Hospital of the World.

Well may it be said of this institution:

> The deeds of charity we have done stay with us forever. Only the wealth we so bestowed do we keep; the other is not ours.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE little book[1] by Clapp on cataract is a timely and useful one, since there had not appeared in English any summary of the recent important advances in our knowledge of the crystalline lens comparable to the book of Siegrist or chapters of Jess and Hess in the larger German handbooks.

The chapters contributed by Ida Mann on the embryology and comparative anatomy of the lens are excellent for the amount of space given them and are especially important for the study of this fascinating organ, whose entire history from the closing of the lens vesicle to adult life is now made visible to the clinician by the slit-lamp and corneal microscope. Knowledge of this history allows proper interpretation of the various forms of congenital lens opacity and their distinction from acquired or senile opacities which are progressive. The more important of these congenital forms are described and illustrated, in some instances by good original drawings from the Wilmer Institute. In most cases, illustrations have been carefully selected from the previous literature.

A chapter on the chemistry of the normal and pathological lens shows a thorough knowledge of recent important work, including the demonstration of true oxygen metabolism in the avascular lens and of the presence of glutathione as the important hydrogen donator in this process. This substance is absent in the completely cataractous lens, and this fact is possibly of importance in the genesis of cataract. In spite of the great amount of chemical investigation, and a knowledge of the etiology of certain types of cataract such as that after removal of the parathyroids, no generally accepted theory as to the etiology of senile cataract has been formulated. The author's own investigation of the growth of the lens and its increase in weight from the seventh to the one hundred and fiftieth day is interesting.

In discussing the various types of acquired cataract and their treatment the author includes a great number of references to the literature, but at times has apparently not fully digested this work, since work of little importance is given equal space with that of real value. Thus it was hardly necessary to mention the injection of nitrogen into the anterior chamber to prevent traumatic cataract, a procedure purely experimental and clinically untried while the brief assembly of positive and negative evidence as to the occurrence of cataract in leprosy leaves us practically where we started. The classification of senile cataract is not very clear, since surely "peripheral cataracts" and "cortical opacities" are one and the same, while "morgagnian cataract" is definitely a subdivision of "hypermature cataract."

The non-surgical treatment of cataract is discussed, as it should be, without enthusiasm, though perhaps the author's negative conclusions might be even more strongly stated.

The discussion of the various types of cataract operation, anæsthesia, paralysis of the lids before operation (akinesia), and after-care, is excellent. In discussing Verhoeff's method of intracapsular extraction, no mention is made of an important change in his technique by which the lens capsule is grasped at the upper equator instead of below. In Sinclair's method, the important fact that his capsule forceps are made with a cross-action handle, and hence retain their hold on the capsule automatically, is omitted. It seems to the reviewer that among the methods of operating for glaucoma following cataract extraction, cyclo-dialysis, which is employed as the operation of choice by Elschnig and others for this condition, should have been included.

The discussion of the complications of the cataract and their treatment is quite complete. It is only natural that the theory of anaphylactic reaction to lens protein as an explanation of reactions following retention of cortex should be accepted by the author, on account of the work of his associates, although many authorities differ with him on this point. The full bibliographies offer an excellent opportunity to those interested in further study of particular questions which arise; altogether the book is well adapted to stimulate such study.

SANFORD R. GIFFORD.

THE appearance of the second edition of *Practical Obstetrics*[2] by Bland and Montgomery within 2 years is proof of the popularity of the first edition. The present edition is an improvement over its predecessor but a few points of difference may be noted between the authors and the reviewer. In the differential diagnosis between simple vomiting of pregnancy and pernicious vomiting, the authors say (p. 159) that the "neurotic element (is) lacking" in the latter. This statement is at variance with the opinion of many obstetricians. In the discussion of

[1] CATARACT; ITS ETIOLOGY AND TREATMENT. By Clyde A. Clapp, M.D., F.A.C.S. Philadelphia: Lea & Febiger, 1934.

[2] PRACTICAL OBSTETRICS FOR STUDENTS AND PRACTITIONERS. By P. Brooke Bland, M.D., and Thaddeus L. Montgomery, M.D. 2d ed. Philadelphia: F. A. Davis Co., 1934.

the etiology of eclampsia (p. 173), nothing is said of the possible pituitary origin of this obstetric complication. Since Hofbauer first called attention to the important rôle played by the pituitary gland in eclampsia, valuable contributions on this subject have appeared in the literature, notably by Anselmino and Hoffmann and by Fauvet. There is very much to be said in favor of a pituitary origin of eclampsia. In dealing with the management of labor in eclamptic patients no mention is made of local anæsthesia, in spite of the fact that this form of anæsthesia is far safer than general anæsthetics for women who have toxæmia. The authors evidently have very little regard for local, infiltration anæsthesia in obstetrics because in the chapter on anæsthesia (p. 493), only four lines are devoted to this type of anæsthesia. The reviewer is firmly convinced that both the maternal mortality and morbidity can be considerably reduced by the more frequent use of local infiltration anæsthesia particularly for women who have toxæmia, nephritis, pulmonary complications, pronounced anæmia, and other complications. However, spinal anæsthesia for obstetric operations is properly condemned by the authors. As a contrast to the space allotted to local anæsthesia, four pages and four illustrations are devoted to inversion of the uterus, which is a rare complication (p. 538). The authors speak of the examination made about 6 weeks after delivery as the "final examination" (p. 489). Even though they say that subsequent postnatal visits should be arranged where necessary, it is best to avoid the use of the words "final examination." Many women interpret this to mean the final contact with their physicians until a new pregnancy occurs; but all of us including the authors are attempting to educate women to visit their physicians for at least one examination a year.

In spite of the foregoing minor differences, the book is one of the best on the subject of obstetrics. The text is written in clear language, the illustrations are abundant and instructive, and the typography is excellent. Both the authors and the publisher are to be congratulated. J. P. GREENHILL.

THE new ninth edition of Davis' *Applied Anatomy*[1] conforms quite closely to the previous editions with which the reviewer is familiar and, in particular, to the sixth edition with which it was carefully compared. It is indeed a tribute to Dr. Muller that so few changes have been necessary in this present edition. These changes have been made particularly in those sections dealing with the brain and meninges, the female pelvis and perineum, the prostate and male perineum and external genitalia. A few added paragraphs here and there attest to the value of the older edition. The present volume contains 717 pages as against 638 pages in the sixth edition. There are about 45 new illustrations and many of the older illustrations are redrawn. Some of the plates are beginning to show the effects of frequent printing. It is to be regretted that the book is printed on such heavy paper stock which, though of excellent quality, adds considerable to the bulk of the volume and makes it a little inconvenient to read and handle. MICHAEL L. MASON.

[1] APPLIED ANATOMY; THE CONSTRUCTION OF THE HUMAN BODY CONSIDERED IN RELATION TO ITS FUNCTIONS, DISEASES, AND INJURIES. By Gwilym G. Davis, M.D. 9th ed. revised by George P. Muller, M.D. Philadelphia, London, and Montreal: J. B. Lippincott Company, 1934.

THE various opinions found in current literature on carcinoma of the cervix, uterine endometrium, and vulva are thoroughly reviewed, but many of questionable value are emphasized, by Meigs in *Tumors of the Female Pelvis*.[2] The author's tendency toward surgical procedures is apparent and may lead some of his readers to attempt operations which their experience and training do not justify. His description of irradiation therapy is excellent.

Too many classifications of ovarian neoplasms have been offered in textbooks to warrant another which varies so little in fundamentals from the two after which it is patterned. The descriptions of these tumors are excellent, the substance of the entire chapter well arranged, and the treatment thoroughly sound.

The chapter on endometrioma presents clearly the theories of origin of these interesting tumors. The drawings and subject matter outline clearly the variations in their behavior. His treatment is probably more radical than that recommended by many recent contributors.

Any discussion of chorioma is difficult to present on account of the paucity of material and the uncertainty of our information on the behavior of these trophoblastic cells. This necessitates reference to those classic reports in the literature, among which there is such a wide difference of opinion.

In general this monograph represents an enormous amount of painstaking labor; the drawings and photomicrographs are excellent. Some chapters reflect hasty compilation and editing.

The deductions made from the statistical study of a limited amount of material are not convincing.

H. O. JONES.

THE two volume work of Romanis and Mitchiner on *The Science and Practice of Surgery*[3] has reached its fifth edition in 7 years which speaks for itself as to its reception by the profession.

The new edition has some minor changes in the chapters on peritonitis, fractures, burns, and varicose veins. In most instances these are agreeably acceptable and show that the authors are making an effort to keep the volumes abreast of the changes that are constantly taking place.

There have also been alterations and additions in the chapters on eye, ear, nose, and throat and in

[2] TUMORS OF THE FEMALE PELVIC ORGANS. By Joe Vincent Meigs, M.D., F.A.C.S. With a foreword by Robert B. Greenough, M.D. New York: The Macmillan Co., 1934.

[3] THE SCIENCE AND PRACTICE OF SURGERY. By W. H. C. Romanis, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), and Philip H. Mitchiner, M.D., M.S. (Lond.), F.R.C.S. (Eng.). 5th ed. vol. I, General Surgery; vol. II, Regional Surgery. Philadelphia: Lea and Febiger, 1934.

the chapter on anæsthetics. A few new illustrations have been added and some of the old ones deleted.

The reviewer is of the impression that the popularity of this work will continue because of its general excellence as a compact textbook and reference work.

R. W. McNEALY.

FOR more than thirty years, Dr. Craig, the author of *Amebiasis and Amebic Dysentery*,[1] made a careful study of amœbic infection from the laboratory, epidemiological, and clinical standpoints. He has combined his experience with a survey of the most important data on this subject that has accumulated during the past two decades. Probably no man is more capable of presenting what is considered the modern interpretation of amœbiasis and amœbic dysentery than this author.

During the past several years many important advances have been made in our knowledge of the etiology, epidemiology, prophylaxis, and treatment of this infection. While the literature on the subject has been tremendous, most of it has been published in research medical journals that do not reach the hands of most practicing physicians.

The epidemic of amœbic dysentery which originated in Chicago in 1933, which resulted in over 800 cases of the disease, has awakened intense interest in amœbiasis among the physicians of this country. In 1911, 10 years after Craig published his first paper on amœbiasis, Sistrunk first called attention to the frequent presence of Endamœba histolytica in people in the northern part of the United States. The author calls attention to the fact that approximately 5 to 10 per cent of the people of this country harbor this parasite.

Craig's book is particularly timely as no work has appeared for a number of years which has been devoted exclusively to amœbiasis. It contains 315 pages and the contents are divided into 12 chapters discussing the etiology, epidemiology, pathology, symptomatology, complications and sequelæ, the clinical and laboratory diagnosis, including the complement fixation test, the prognosis, prophylaxis, and treatment. The illustrations throughout the book are exceptionally good.

The chapter on prognosis is very enlightening and calls attention to the fact that with the present methods of treatment excellent prognosis can be anticipated. In individuals in whom the infection is not recognized or who are improperly treated, the prognosis is unfavorable as to continued health but favorable so far as actual danger to life is concerned. If we assume that from 5 to 10 per cent of the population of the United States are infected with Endamœba histolytica and then consider the comparatively small number of deaths from this infection in the United States as recorded in the health statistics, it would appear that the prognosis of amœbiasis is extremely favorable, so far as danger to life is concerned, and that the mortality is extremely low. The prognosis in amœbic abscess of the liver and lung is always grave even with modern methods of treatment.

Extremely valuable data regarding prophlaxis is presented, including a clear conception of the methods of transmission of this parasite, its resistance to chemical and physical agents, the proper disposal and treatment of fæces, protection and sterilization of water supplies, protection of food from flies, the detection and treatment of carriers, education of the public, personal hygiene, and the importance of public health problems.

There can be no doubt that this is the last word on the subject of amœbiasis and amœbic dysentery. It is masterfully handled and undoubtedly will be a great aid in the diagnosis, prophylaxis, and treatment of amœbiasis. The author has without question presented material that will disseminate proper knowledge to the medical profession with unlimited value to the general practitioner and to the public health officers.

CLIFFORD J. BARBORKA.

SINCE 1927 when Calmette and Guerin first started using their attenuated bovine tubercle bacillus in vaccinating infants, a large and controversial literature has developed concerning it. Work has been done and articles published proving that it is and is not dangerous and that it does and does not provide immunity. The book[2] under consideration is extremely valuable in that it presents briefly a summary of this contradictory literature and sets forth the case for and against the vaccine fairly and judiciously. As a result of his investigations the author concludes:

1. The B.C.G. is not a *virus fixe* but can (or could until recently) increase in virulence when suitably grown.

2. The B.C.G. does occasionally produce progressive tuberculosis in laboratory animals.

3. The B.C.G. has not yet been proved to have caused progressive tuberculosis in man.

4. A definite degree of immunity is produced in cattle by the B.C.G. vaccine; in other animals the results are not so clear.

5. A certain increase of immunity is produced in man by the B.C.G. vaccine, but it is impossible to say yet whether enough protection is produced to warrant the reorganization of our present scheme for dealing with tuberculosis.

6. Research should be begun in England to decide the degree of immunity produced in man, but in the meantime the B.C.G. vaccine should be given to children in tuberculous families to supplement the existing measures.

JEROME R. HEAD.

A COMPREHENSIVE treatise on certain types of pathological changes about the jaws is presented by Dr. Thoma in a book[3] containing 630

[1] AMEBIASIS AND AMEBIC DYSENTERY. By Charles F. Craig, M.D., M.A., F.A.C.S., F.A.C.P. Springfield, Illinois, and Baltimore, Maryland: Charles C. Thomas, 1934.

[2] The B. C. G. VACCINE. By K. Neville Irvine, D.M., M.A., B.Ch. (Oxon.) M.R.C.S., L.R.C.P. London: Oxford University Press, 1934.

[3] CLINICAL PATHOLOGY OF THE JAWS; WITH A HISTOLOGICAL AND ROENTGEN STUDY OF PRACTICAL CASES. By Kurt H. Thoma, D.M.D. Springfield, Illinois, and Baltimore, Maryland: Charles C. Thomas, 1934.

pages and 400 illustrations. The work is replete with case reports illustrated by slides, photographs, and roentgenograms. The remainder of the text is concerned with pathogenesis, microscopic and roentgen findings, rather than clinical diagnosis and prognosis. Practically nothing is given on clinical treatment except in case reports.

Malformations, fractures, and inflammation of the jaws are treated adequately, and endocrine and nutritional disturbances are each given a chapter. At least two-thirds of the book is allotted to cysts and tumors which are discussed exhaustively and very lucidly. The thoroughness of preparation is suggested in the unnecessarily complete bibliography which is appended to each chapter.

This book is scarcely styled for a textbook but as a reference work it will be a distinct addition to the literature of the subject. CHARLES W. FREEMAN.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

COLLECTION DES INITIATIONS MÉDICALES. Published under the direction of Dr. A. Sézary. Propédeutique Obstétricale. By L. Devraigne. Paris: Masson et Cie, 1934.

COLLECTION DES INITIATIONS MÉDICALES. Published under the direction of Dr. A. Sézary. La Pratique Obstétricale. By L. Devraigne. Paris: Masson et Cie, 1935.

TRANSACTIONS OF THE PACIFIC COAST SOCIETY OF OBSTETRICS AND GYNECOLOGY. Volumes 1, 2, and 3. For the Years 1931, 1932, and 1933. Edited by Albert Mathieu, M.D. Portland, Oregon: The Western Journal of Surgery, 1934.

THE CARE OF THE AGED, THE DYING AND THE DEAD. By Alfred Worcester, M.D., Sc.D. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1935.

SURGICAL DISEASES OF THE CHEST. By Evarts Ambrose Graham, A.B., M.D., F.A.C.S., Jacob Jesse Singer, M.D., F.A.C.P., and Harry C. Ballon, M.D., C.M., F.A.C.S. Philadelphia: Lea & Febiger, 1935.

BLOOD GROUPS AND BLOOD TRANSFUSION. By Alexander S. Wiener, A.B., M.D. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1935.

PARENTHOOD: DESIGN OR ACCIDENT? A MANUAL OF BIRTH-CONTROL. By Michael Fielding, M.D. Preface by H. G. Wells. New York: The Vanguard Press, 1935.

THE CYCLOPEDIA OF MEDICINE. Edited by George Morris Piersol, B.S., M.D., assisted by Edward L. Bortz, A.B., M.D. Index to Vols. 1 to 12. Philadelphia: F. A. Davis Co., 1934.

THE NERVOUS PATIENT; A FRONTIER OF INTERNAL MEDICINE. By Charles Phillips Emerson, M.D. Philadelphia, London, and Montreal: J. B. Lippincott Co., 1935.

THE ROMANCE OF EXPLORATION AND EMERGENCY FIRST-AID FROM STANLEY TO BYRD. New York: Burroughs Wellcome & Co., 1934.

THE GLASGOW ROYAL MATERNITY AND WOMEN'S HOSPITAL. Medical Report for the Year 1933. Prepared by D. McK. Hart, M.B., Ch.B., F.R.F.P.S.G., M.C.O.G. Glasgow: Aird & Coghill, Ltd., 1934.

HUGH OWEN THOMAS; A PERSONAL STUDY. By Frederick Watson. London: Oxford University Press, 1934.

A MANUAL OF OBSTETRICAL AND GYNÆCOLOGICAL PATHOLOGY. By John H. Teacher, M.D. Edited by Alice J. Marshall, M.B. London: Oxford University Press, 1935.

ANÆSTHESIA AND ANALGESIA IN LABOUR. By Katharine G. Lloyd-Williams, M.D., B.S. (Lond.). With a Foreword by Dame Louise McIlroy, D.B.E., D.Sc., M.D. (Glasg.), D.Sc. (Lond.), D.Sc., M.R.C.P., F.C.O.G. Baltimore: William Wood & Co., 1934.

HOEBER'S SURGICAL MONOGRAPHS. CORRECTIVE RHINOPLASTIC SURGERY. By Joseph Safian, M.D. New York: Paul B. Hoeber, Inc., 1935.

OXYGEN AND CARBON DIOXIDE THERAPY. By Argyll Campbell, M.D., D.Sc. (Edin.), and E. P. Poulton, M.A., D.M. (Oxon.), F.R.C.P. (Lond.). Foreword by Sir Leonard Hill, F.R.S. London: Oxford University Press, 1934.

THE PRACTITIONERS LIBRARY OF MEDICINE AND SURGERY. Volume 8—Therapeutics. New York and London: D. Appleton-Century Co., 1935.

BIOLOGÍA Y PATOLOGÍA DE LA MUJER; TRATADO DE OBSTETRICIA Y GINECOLOGÍA, PUBLICADO BAJO LA DIRECCIÓN DE LOS DOCTORES JOSEF HALBAN Y LUDWIG SEITZ. Traducido directamente del original alemán por Joaquín Núnez Grimaldos, con la colaboración técnica del Dr. D. Arcadio Sánchez López. Tomo 15. Madrid: Editorial Plus Ultra, 1935.

PHYSIOLOGIE UND PATHOLOGIE DER WEHEN; ERGEBNISSE DER WEHENMESSUNG. By Dr. Tassilo Antoine. Vienna: Wilhelm Maudrich, 1935.

NAMES OF SURGICAL OPERATIONS; COMPILED AND ARRANGED BY THE WESTERN SURGICAL ASSOCIATION THROUGH ITS SPECIAL COMMITTEE. Edited by Carl E. Black, A.M., M.D. St. Paul Minn · Bruce Publishing Co., 1935